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SCREEN INTENSIFICATION SYSTEMS AND THEIR LIMITATIONS*†

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I. INTRODUCTION

IN RECENT years, there has developed among roentgenologists a considerable interest in methods by which the brightness of roentgenoscopic screens may be intensified. This interest has been aroused principally by the relatively poor detail provided by present-day roentgenoscopic screens and by the belief that this detail might be materially improved if screen brightness were increased by a factor of several thousand times. The basis for this belief may be found in the investigations of Chamberlain[2] and Hecht.[6] Chamberlain has demonstrated that roentgenograms made with roentgenoscopic screens in contact with roentgenographic films exhibit much more detail than the same screens when observed at roentgenoscopy; and Hecht has shown that the human eye when viewing fields having a luminance approaching that encountered in roentgenoscopy (10^{-4} to 10^{-2} foot-lambert) has a visual acuity or ability to perceive detail, of only a small fraction of that occurring under normal lighting conditions (10 to 100 foot-lamberts). Thus it appears that the poor performance of the roentgenoscope may be attributed to deficiencies of the human eye when operating at the low levels of illumination which occur during roentgenoscopy rather than to any inherent imperfections in the roentgenoscope itself and that this performance should be improved if the brightness of the screen were increased to levels at which the eye performs more satisfactorily.

As convincing as the foregoing evidence may be the premise that an improvement in roentgenoscopic clarity will surely follow an increase in the brightness of the roentgenoscopic screen should not be wholly accepted until it is certain that the causes of poor visual acuity which occurs at low levels of illumination will not also influence the performance of any man-made optical device which may be used to intensify screen brightness. It is entirely possible that the limitations of the human eye are caused by factors which also limit all types of optical systems, physical as well as physiologic. If this is the case the use of an

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intensifying device in conjunction with a roentgenoscope may cause little if any improvement in screen clarity.

Rose[11] has recently shown that the performance of the eye at any level of illumination is largely limited by statistical fluctuations in the number of light photons reaching the retina during the observation of an event. Although most individuals are completely unaware that such fluctuations exist, they nevertheless are present to a greater or lesser degree under all viewing conditions and ultimately determine the fineness of structure that can be perceived. They of course do not constitute the only factor which limits visual performance. Such factors as the diameters of the retinal rods and cones also introduce limitations, but, as Rose has demonstrated, these factors only contract the limits already set by statistical theory.

In general, the statistical fluctuations in light intensity which occur at the retina are proportional to the square root of the average number of photons projected on the retina within the storage time of the eye (about 0.2 second). For example, if the eye is observing an object, let us say 1 mm. in diameter at a viewing distance of 20 cm., the number of photons reaching the retina from the object during each 0.2 second will be of the order of one million at normal levels of illumination. Therefore, the average fluctuation in light intensity at the retina from moment to moment will be approximately 1,000 photons or 0.1 per cent of the average light intensity. Now, if the luminance of the object is reduced to levels comparable to that occurring at roentgenoscopy, the effective number of photons reaching the retina within the storage time of the eye may be as small as one hundred. Accordingly, such a number will have an associated average fluctuation of 10 photons or 10 per cent of the average light intensity. Thus when the luminance of an object is reduced, the per cent fluctuation in the rate at which photons arrive at the retina increases.

The effect of this fluctuation will be evident if one considers the situation that might occur if the 1 mm. object under observation is surrounded by a field having an average luminance 5 per cent greater or less than that of the object; that is, the contrast between the object and the surrounding field is 5 per cent. Under such a condition, if the object is observed under normal conditions of illumination, the 0.1 per cent fluctuation in the light intensity at the retina will not interfere noticeably with the perception of the object. On the other hand, if the luminance of the object is at a level corresponding to that occurring at roentgenoscopy where the fluctuation is of the order of 10 per cent, the object may at one moment appear darker and at the next moment lighter than the surrounding field. Obviously such variation will interfere markedly with the perception of the object.

It will be apparent from the manner in which statistical fluctuations in light intensity influence visual performance that these same fluctuations will also have a profound effect on the ability of any optical system, including roentgenoscopic screen intensifying devices, to record detail. It therefore will be wise for us to investigate carefully the magnitude of this effect before we raise our hopes too high in anticipation of the results that may be achieved with screen intensification.

II. FLUCTUATION THEORY APPLIED TO ROENTGENOSCOPY

It may be difficult for some to understand the bearing which statistical fluctuation theory has on the performance of the roentgenoscope. However, one or two simple examples should quickly clarify this trouble.

If one takes from his pocket a coin and tosses it a large number times, one may expect the coil to fall *heads* uppermost 50 per cent of the time and *tails* uppermost the remaining 50 per cent of the time. Now, if instead of a large number of tosses one makes, let us say, 100 throws, it will be rather unusual to obtain exactly 50 heads

and 50 tails. Instead, one may perhaps find 58 heads and 42 tails. Also, if the experiment is repeated again and again, the result on the second try may be 47 heads and 53 tails, on the third try, 44 heads and 56 tails, and so on. Although one may expect to obtain on the average an equal distribution of heads and tails, there will be in any given series of throws of the coin a certain amount of fluctuation about that average due to the random nature in which the coin is tossed.

The situation which occurs during roentgenoscopy is not so very different from that prevailing in the coin-tossing experiment. In roentgenoscopy, roentgen-ray photons are projected through the various anatomical structures under examination. Under usual roentgenoscopic conditions, the number of photons is of the order of 10 million per second for each square millimeter of exposed surface of the patient's body. If it is the abdomen that is under examination about 1 per cent or 100,000 of these photons each second emerge on the average to fall on the roentgenoscopic screen. Now the absorption of roentgen-ray photons within the human body is almost surely a completely random phenomenon; that is, there is a certain probability that the tissues will absorb a given photon but there is no certainty that they will do so. Therefore, if one actually measures the number of photons emerging from each square millimeter of the abdomen each second, one may find 102,000 photons emerging during the first second, 99,000 during the second second, and so on. Or if one takes measurements simultaneously over several similar areas of the abdomen, the readings will fluctuate from one region to another just as the readings fluctuate from time to time within a single region.

Although the knowledge that the number of photons which comprise a roentgen-ray beam fluctuates from instant to instant is interesting, it is of little practical value unless the magnitude of the fluctuations can be determined. In order to derive the formulae by which such calculations may be made, let us return for a moment to the coin-tossing experiment previously cited. We have seen that when the coin is tossed many times the probability of throwing heads (or tails) is 50 per cent, but that when the coin is tossed a relatively few times, say 100, the number of heads may be only 42; that is, the result shows a deviation from the expected yield. Now if one repeats the experiment of throwing the coin 100 times over and over and on each occasion observes the number of heads that are thrown, a wide variety of values will be obtained that range on either side of the average value of 50. Now one might calculate the average fluctuation of the results of the experiment by adding the various deviations from the expected yield and dividing the total by the number of observations. However, since the number and magnitude of minus deviations below 50 are approximately equal to the number and magnitude of plus deviations above 50, the resultant average would approach zero and therefore give a completely erroneous impression of the size of the fluctuation. For this reason it is conventional statistical practice when calculating average fluctuation to take the various deviations and square them (multiply them by themselves). Under this circumstance, all values become positive. The values are then averaged by adding them together and dividing the total by the number of observations. This result, of course, gives a squared value of the average fluctuation which may be reduced to standard terms by taking its square root.

The value of the average fluctuation derived by this method has been termed by statisticians *the standard deviation*. It is an excellent index of the magnitude of the fluctuations occurring in a random phenomenon and accordingly its use for this purpose has found wide application. Whenever the term standard deviation is used hereafter it will refer to the root mean square value of the average fluctuation as determined by the method just cited.

It may be easily shown by experiment

and statistical theory that the standard deviation or average fluctuation which occurs in a random phenomenon may be calculated from the equation

$$s = \pm\sqrt{npq} \qquad (1)$$

where s is the standard deviation,
n is the number of events occurring in the phenomenon,
p is the probability of an event turning out positively, and
q is the probability of an event turning out negatively.

Now in the coin-tossing experiment, the number of throws in each part of the experiment was 100, and therefore in calculating the average fluctuation of the occurrence of heads from equation (1), such a number should be substituted for the symbol n. Also, the probabilities of a coin turning heads and tails are each equal to $\frac{1}{2}$; therefore, p and q in equation (1) have values of 0.5. Thus the average fluctuation in the number of heads should be

$$s = \pm\sqrt{100 \times 0.5 \times 0.5} = \pm 5. \qquad (1A)$$

Actually if the experiment is performed, a value very close to 5 will be obtained.

In most roentgen phenomena, the probability of an event turning out positively is rather small. For example, in the roentgen beam projected through a patient's abdomen the probability of an incident photon emerging from the patient's other side is about 1 per cent; that is, there is a 99 per cent chance that the photon will be absorbed or deviated from its course, never to emerge as an image-forming photon. Therefore, under such circumstances, the value of q in equation (1) approaches a value of 1.0 and equation (1) may be written

$$s = \pm\sqrt{np} \qquad (2)$$

where n is the number of roentgen-ray photons entering the roentgen phenomenon.

However, np is obviously the number of photons emerging from the phenomenon; therefore,

$$s = \pm\sqrt{n_e} \qquad (3)$$

where n_e is the number of emergent photons.

It will be recalled that several paragraphs ago we stated that a typical emergent abdominal roentgenoscopic beam has an average yield of approximately 100,000 photons per second per square millimeter of presenting surface. It will be observed that equation (3) indicates that the average fluctuation associated with such a beam will be 315 photons/sec/sq. mm. or 0.3 per cent.

It may be easily shown that equation (3) is valid whether one speaks of roentgen-ray photons in a roentgen beam, light photons in a light beam, electrons in an electrical system, and so forth. It is the fundamental statistical equation indicating the magnitude of the fluctuations occurring in any random process where the chances of success are small.

Until now, we have considered the fluctuations which occur in simple random phenomena where only a single factor contributed to the deviations. However, in the roentgenoscopic process, fluctuations occur at several different phases of the process and accordingly the simple formula presented in equation (3) is not applicable. In the roentgenoscopic process, the sources of fluctuation are numerous. We have seen how fluctuations occur during the absorption of the roentgen beam in the patient's tissues. Similar fluctuations also occur in the absorption of the beam in the roentgenoscopic screen. Fluctuations arise, too, in the roentgen tube itself. The liberation of electrons from the tube's filament is a random process, and accordingly there will be deviations in the rate at which electrons arrive at the target and produce roentgen rays. Then, too, the production of roentgen rays by electron impacts on the target is a random process; that is, there is a certain probability that an electron will produce a roentgen-ray photon, but there is no certainty that it will do so. Therefore, fluctuations will arise at the instant that roentgen rays are formed. Additional sources of fluctuation occur in the production of light photons by roentgen-ray photons in the

roentgenoscopic screen and in the production of nerve impulses when the light photons fall on the retina. Furthermore, when a screen intensifier is placed in the system several sources of fluctuation may be introduced by the device.

Now these sources of fluctuation in the roentgenoscopic process are essentially independent of one another; that is, the absorption of roentgen-ray photons, for example, in the tissues of the patient has no bearing on the efficiency of roentgen-ray photon production in the roentgen tube. Therefore, it may be easily shown by statistical theory that the average total fluctuation at the termination of the roentgenoscopic process when the nerve impulses are finally transmitted from the retina to the brain is given by the equation

$$s^2 = s_1^2 + s_2^2 + \cdots s_n^2 \qquad (4)$$

where $s_1, s_2, \cdots, s_n$ are the values at the retina of the various average fluctuations introduced at the various phases of the roentgenoscopic process.

Now the fluctuation introduced at any particular phase of the process is given by equation (3); that is,

$$s_{x0} = \pm n_x^{1/2} \qquad (5)$$

where s_{x0} is the fluctuation at the level of the x phase of the roentgenoscopic process and

n_x is the number of photons or electrons, as the case may be, observed at that phase.

This fluctuation, of course, will become greater or less as it proceeds through the roentgenoscopic process depending on whether the succeeding stages of the process increase or decrease the process' intensity. For example, the fluctuations produced in the roentgen beam during its passage through an anatomical structure will diminish in magnitude as the beam is absorbed by the roentgenoscopic screen since only a fraction of the beam undergoes screen absorption. Immediately thereafter, however, it will increase in magnitude as each absorbed roentgen-ray photon produces many light photons and increases the intensity of the process. Now if g_x is the amplification factor through which the roentgen process passes between the x phase and its final phase within the observer's retina then

$$s_x = s_{x0} g_x = \pm g_x n_x^{1/2} \qquad (6)$$

where s_x is the value of the retina of the average fluctuation introduced at the x phase of the process.

Equation (6) represents the general expression for each of the terms in equation (4); therefore, equation (4) may be written

$$s^2 = g_1^2 n_1 + g_2^2 n_2 + \cdots g_n^2 n_n. \qquad (7)$$

But by definition of the term, g,

$$n_1 g_1 = n_2 g_2 = \cdots n_n g_n = n_n. \qquad (8)$$

Therefore,

$$s^2 = n_n^2 \left(\frac{1}{n_1} + \frac{1}{n_2} + \cdots \frac{1}{n_n} \right). \qquad (9)$$

It will be evident from equation (9) that the value of s will largely be determined by the term which has the smallest value of n. Therefore, equation (9) as a first approximation may be written:

$$s^2 = n_n^2 / n_a \qquad (10)$$

where n_a is the smallest value to which the number of photons (or electrons) descends in the roentgenoscopic process.

However,

$$n_a g = n_n \qquad (11)$$

where g is the amplification factor through which the roentgen process passes between the stage of n_a to its final stage in the observer's retina, therefore

$$s = \pm g^{1/2} n_n^{1/2}. \qquad (12)$$

When a quantity of photons, let us say n_n, from an object under observation arrives at and stimulates the retina of an observer within the storage time of the eye, the object will be seen if the number of photons is greater or less by a certain minimum amount than the number of photons

stimulating the retina from the visual field surrounding the object. Rose[11] has found that this minimum difference is proportional to the average fluctuation, s, of the number of stimulating photons from the object; that is,

$$\Delta n = ks = \pm kg^{1/2} n_n^{1/2} \tag{13}$$

where Δn is the smallest difference between the number of photons in the retinal image and its surrounding field which allows perception of the image and k is a proportionality constant, which may be conveniently termed the threshold *contrast to fluctuation ratio.*

Now the contrast of an image is usually defined as the ratio of the difference in light intensity between an image and its surrounding field to the light intensity of the image. Therefore the per cent threshold contrast for image perception is given by the equation

$$C = \frac{\Delta n}{n_n} \times 100 \cdot \tag{14}$$

When n is eliminated from equations (13) and (14)

$$C^2 = \frac{k^2 g}{n_n} \times 10^4 \cdot \tag{15}$$

Now if n_r is the number of stimulating photons which reach the retina from the object each second, then

$$n_r t = n_n \tag{16}$$

where t is the storage time of the eye.

Also, if n_0 is the number of stimulating photons which reach the retina from each square millimeter of the roentgenoscopic screen or pick-up surface of the screen intensifier if such a device is used,

$$n_r = \frac{\pi \delta^2 n_0}{4} \tag{17}$$

where δ is the diameter of the object under observation.

Therefore

$$C^2 = \frac{4k^2 g}{\pi \delta^2 t n_0} \times 10^4 \tag{18}$$

or

$$\delta = \pm \frac{2k}{C} \left(\frac{g}{\pi t n_0} \right)^{1/2} \times 10^2. \tag{19}$$

Equation (19) specifies the size, δ, of the smallest discernible object and its contrast, C, in terms of (1) the number of stimulating photons, n_0, which reach the retina each second from each square millimeter of the roentgenoscopic screen or pick-up device, (2) the amplification factor, g, of the roentgenoscopic process between the stage of lowest intensity and the stage at which the retina is finally stimulated, (3) the storage time of the eye, t, and (4) the threshold contrast to fluctuation ratio, k, which the eye will tolerate. It will be seen that when contrast increases the minimum object size that can be perceived becomes smaller; that is, the greater the contrast, the finer the detail that may be seen. Also, when the number of photons stimulating the retina (i.e. the brightness of the scene of observation) becomes greater, the size of the smallest perceptible object diminishes in size. These are of course facts that are well known through experience to everyone.

III. Visual Performance during Conventional Roentgenoscopy

If values for the various parameters listed in equation (19) may be obtained for a particular roentgenoscopic system, the ability of that system to record detail at various contrast levels may easily be evaluated. During the past few months we have undertaken in our laboratory a series of investigations by which these parameters have been determined for simple roentgenoscopy, for roentgenoscopy aided by an ideal screen intensifying device, and finally for roentgenography. The values for roentgenography were obtained so that a comparison of the anticipated performance of the ideal roentgenoscopic intensifying system might be made with roentgenographic films. One so often hears the statement that by screen intensification roentgenoscopy may be expected to approach roentgenography in clarity that we deemed it desir-

able to calculate the comparative merits of the two methods.

The first system studied was simple roentgenoscopy itself. The values of g and n_0 in equation (19) were obtained by tracing from the beginning the various phenomena which occur during the roentgenoscopic process.

In simple roentgenoscopy it is customary to operate the roentgen tube at a potential of about 70 kvp., a tube current of 5 milliamperes and with a tube-screen distance of about 27 inches. Also, a filter of 1 mm. of aluminum is usually placed in the roentgen beam.

The number of roentgen-ray photons which are produced under these conditions and which are projected each second toward each square millimeter of the roentgenoscopic screen may be measured in a number of ways. The method which we employed was that in which the number of roentgens per second, produced by the beam at a distance of 27 inches from the tube's target, was measured and applied to the well-known conversion formula

$$N=\frac{32r}{eV\rho u/\rho}\times 10^{-2} \qquad (20)$$

where N is the number of roentgen-ray photons per second projected on each square millimeter of screen surface,
r is the experimentally measured number of roentgens,
e is the charge on the electron,
V is the energy in electron volts of each photon,
ρ is the density of air, and
u/ρ is the mass absorption coefficient of air.

This method of measurement has the advantage that, although the energies of the photons in the roentgen-ray beam extend through a considerable range, no tedious mathematical manipulations are incurred in the use of the formula because the product of $V\times u/\rho$ remains relatively independent of photon energy through the range of tube voltages employed in medical roentgenoscopy. This independence is well shown in Figure 1 where it is seen that in the energy range between 25 and 70 kev., (the energy range of the photons in a beam produced by a tube operating at 70 kvp.) the value of the product is $1.1\times 10^4 \pm 10$ per cent.

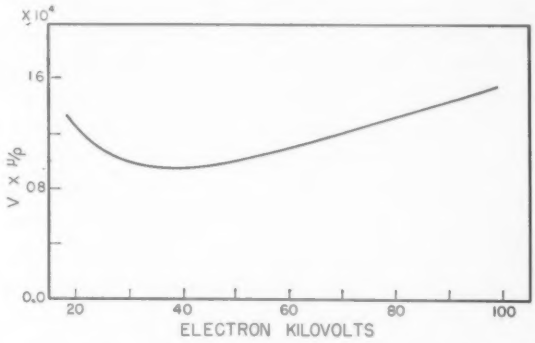

Fig. 1. Curve in which the product of V, the energy in electron volts of a roentgen-ray photon, and u/p, the mass absorption coefficient of air for that photon is plotted as a function photon energy.

The number of roentgens per second measured at the screen site was 0.088 and therefore 5×10^6 photons per square millimeter per second are delivered to the screen by a roentgen tube operating under the average roentgenoscopic conditions under consideration.

Only a small fraction of the photons projected by the roentgen tube toward the fluorescent screen actually reach the screen since most are absorbed by the anatomical structure under examination.[8] For example, the chest transmits approximately 10 per cent of the radiation incident upon it while an anteroposterior projection of the abdomen transmits about 1 per cent; and a lateral abdominal projection transmits the extremely low fraction of approximately 0.1 per cent. Therefore, the number of roentgen-ray photons that finally reach the roentgenoscopic screen is governed by the anatomical structure under observation and amounts to 5×10^5 photons per square millimeter per second for chest roentgenoscopy, 5×10^4 photons per square millimeter per second for anteroposterior abdominal roentgenoscopy, and 5×10^3 pho-

tons per square millimeter per second for lateral abdominal roentgenoscopy.

All of the photons that arrive at the screen surface, of course, do not create luminescence; only those that are absorbed may be expected to do so, and even some of these may not be light-producing for a portion dissipate their energy in the production of characteristic roentgen radiation which subsequently may escape from the screen. The possibility of this occurrence in roentgenoscopic screens, however, is not large because the phosphor commonly used in these screens is zinc sulfide, a material whose characteristic radiations are of such long wavelength that they are readily absorbed before they have a chance to escape from the screen. Therefore one can be reasonably sure that almost none of the radiation absorbed by a roentgenoscopic screen leaves the screen as characteristic roentgen radiation.

The roentgen-ray absorption of a fluoroscopic screen is governed, among other things, by the quality of the exposing radiation. Now previous work by one of us[9] has shown that the radiation quality normally encountered in medical roentgenoscopy and roentgenography extends through the half-value layer range of 1 mm. to 6 mm. of aluminum. For conventional chest fluoroscopy performed at a roentgen tube potential of 70 kvp., the value is approximately 2 mm. of aluminum while for anteroposterior abdominal and lateral abdominal fluoroscopy, the values approach 4 mm. and 6 mm. of aluminum respectively. Absorption measurements made in the laboratory on a Patterson type B-2 screen, the roentgenoscopic screen that is now finding wide use in fluoroscopy, yielded absorption values of 68 per cent, 55 per cent, and 51 per cent at these three radiation qualities. Therefore, the numbers of roentgen-ray photons per second arriving at each square millimeter of the screen's surface and producing illumination are closely 3.5×10^{5}, 2.5×10^{4}, and 2.5×10^{3} for the three types of roentgenoscopy under consideration.

The roentgen-ray photons absorbed by the screen are of a considerably higher energy level than the light photons that they produce. As a result, one may expect the generation of a considerable number of light photons for each roentgen-ray photon absorbed. The number of light photons produced per incident roentgen-ray photon and projected from the front surface of the screen was determined experimentally for the Patterson type B-2 screen by projecting on it from a distance of 27 inches a roentgen beam generated by a roentgen tube operating at 70 kvp., and 5 milliamperes and filtered by 1 mm. of aluminum. The absorption of the screen under these conditions was 0.6. The energy of the light emitted by the screen per square millimeter per second was then measured by a photoelectric spectrophotometer calibrated in microwatts per sq. mm., and the yield of light photons calculated from the well-known equation

$$N=\frac{\lambda W}{hc}\times10^{7} \qquad (21)$$

where N is the photon yield per sq. mm. per second,
W is the energy of the light emitted each second by the screen in watts per square millimeter,
λ is the wavelength of the emitted light,
h is Planck's constant, and
c is the speed of light.

Now the measured energy of the light emitted by the screen per square millimeter per second was 5×10^{-3} microwatts. Since the average wavelength of the light produced by the type B-2 screen is 540 millimicrons (see Fig. 2), the number of light photons emitted by the screen per square millimeter per second during the test was therefore 1.5×10^{10}. If we assume that all roentgen-ray photons absorbed by the screen are utilized in the production of light and if we take the previously calculated number of roentgen-ray photons incident on each square millimeter of the screen as 5×10^{6} per second, then each roentgen-ray photon produces approxi-

mately 5,000 useful light photons. In view of such a yield, the numbers of light photons emitted per square millimeter of screen surface each second during chest, anteroposterior abdominal and lateral abdominal roentgenoscopy are 1.7×10^{9}, 1.3×10^{8}, and 1.2×10^{7} respectively.

The number of these photons which gain admittance to the observer's eyes is governed by the viewing distance, the size of the observer's pupils, and the light intensity distribution function of the screen. Now most roentgen screens approximate a Lambert type of distribution function and

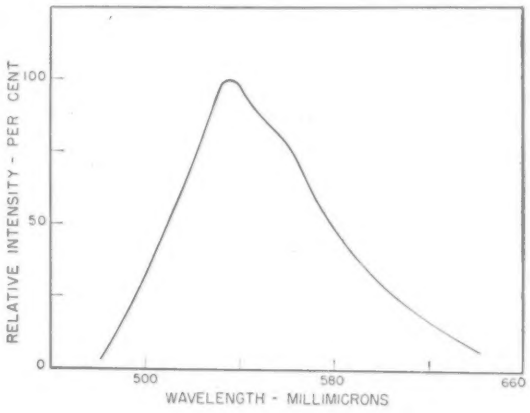

Fig. 2. Spectral emission of the Patterson type B-2 roentgenoscopic screen.

therefore the fraction of the photons that enter the pupil may be shown by simple optical physics to be

$$f=\frac{D^2}{4d^2} \qquad (22)$$

where D is the diameter of the pupil and
d is the viewing distance.

Under the lighting conditions encountered at roentgenoscopy (i.e., 1.7×10^{9} to 1.2×10^{7} photons / sec. / sq. mm. of screen surface), Reeves[10] has shown that the diameter of the pupil ranges from 7.0 to 7.6 mm. (see Fig. 3). Since under customary roentgenoscopic practice, a viewing distance of about 200 mm. is employed, f therefore has a value ranging from 2.5×10^{-4} to 3×10^{-4} and the numbers of photons which gain admittance to the retina from each square millimeter of the screen per second are 5×10^{5}, 3.5×10^{4} and 3.0×10^{3} for the three types of roentgenoscopy previously cited.

Of the photons that gain admittance to the eye, only a small portion is effective in stimulating the retina. Rose has estimated that the number ranges from 0.5 per cent at high levels of illumination to 5.0 per cent at low levels of illumination. These values of

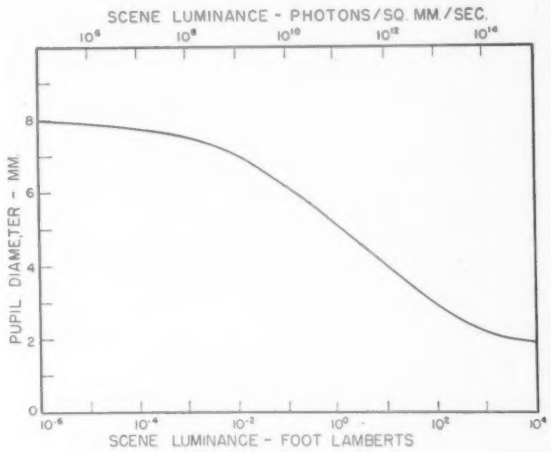

Fig. 3. Relationship between pupil diameter and scene luminance (after Reeves[10]).

retinal efficiency were calculated with the aid of equation (19) from data on visual performance, published by Connor and Ganoung,[5] Cobb and Moss,[3] and Blackwell.[1] In these calculations, Rose employed a value of 0.2 second for the storage time of the eye and a value of 5.0 for the threshold contrast to fluctuation ratio. On the basis of considerable evidence cited by Rose, the value for the storage time of the eye is probably valid within reasonable limits through the complete range of scene luminance encountered in roentgenoscopy and in the observation of roentgenographic films. The value for the threshold contrast to fluctuation ratio was determined experimentally only for high levels of illumination by Rose. Although he is uncertain that this value is correct at low levels of scene luminance, we believe it is significant that his calculated value of retinal efficiency at low illumination, determined from a threshold contrast to fluctuation ratio of 5.0, is closely similar to that of Hecht,[7] obtained

independently by experimental measurements on threshold vision.

Although the available evidence indicates that the efficiency of the retina possibly falls between 0.5 per cent at high levels of illumination and 5.0 per cent at low levels of illumination, this evidence is insufficiently complete to permit the precise evaluation of retinal efficiency at the various levels of scene luminance occurring at roentgenoscopy. It seems reasonable to assume, however, that, for lateral abdominal fluoroscopy where scene luminance is extremely low, the efficiency of the retina will approach its maximum value of 5.0 per cent and that for anteroposterior abdominal and chest fluoroscopy, somewhat lower values, possibly 3.0 and 1.5 per cent respectively, will prevail. If such values are applied to the data previously given for the number of photons which gain admittance to the eye, it appears that about 7.5×10^3, 1.0×10^3 and 1.5×10^2 photons per second reach and stimulate the retina from each square millimeter of screen surface in the three types of roentgenoscopy under consideration. These then, are the values of n_0 for use in equation (19).

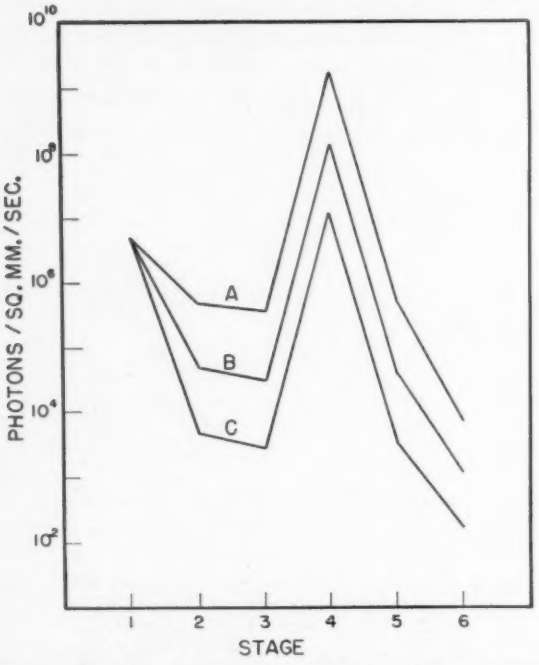

Fig. 4. Graphic representation of the photon densities that exist during the several stages of the roentgenoscopic process. Stage 1: number of roentgen-ray photons projected by roentgen tube toward each square millimeter of screen surface per second; stage 2: number of projected photons remaining after absorption of beam by anatomical tissues; stage 3: number of roentgen-ray photons absorbed by roentgenoscopic screen; stage 4: number of useful light photons produced each second by each square millimeter of screen surface; stage 5: number of light photons which gain admittance to the eye, and stage 6: number of photons admitted to the eye which stimulate the retina. Curve *A* is for roentgenoscopy of the chest, curve *B* for anteroposterior roentgenoscopy of the abdomen, and curve *C* for lateral abdominal roentgenoscopy.

The foregoing series of events which occur in the process of roentgenoscopy is illustrated graphically in Figure 4. There it will be seen that the number of available photons gradually diminishes as the fluoroscopic process progresses until a low point is reached at the stage where roentgen-ray photons are absorbed by the fluoroscopic screen. Thereafter there is a steep rise as roentgen-ray photons are converted to light, and then a long descent at the end of the process when photonic energy finally stimulates the photoreceptor mechanism of the retina. It is evident that the point of lowest intensity within the system is at the final stage of the process. Therefore, for simple roentgenoscopy the value of g in equation (19) is unity. From the values of

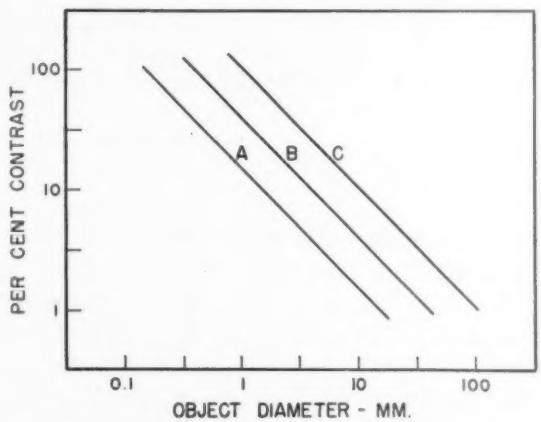

Fig. 5. Theoretical visual performance curves in which percentage threshold contrast is plotted as a function of minimum perceptible object diameter. The designations *A*, *B*, and *C* are for the same conditions given in Figure 4.

n_0, g, t, and k, calculated or cited in the preceding paragraphs, the limits imposed by fluctuation theory on the size and contrast of objects seen at roentgenoscopy may be easily calculated. Curves derived from such calculations in which just perceptible object size is plotted as a function of contrast for chest, anteroposterior abdominal and lateral abdominal roentgenoscopy are shown in Figure 5. Objects having a size and contrast that fall above and to the right of each curve should be readily visible to an observer. Objects having a size and contrast that fall below and to the left of each curve should not be seen. As one might expect, considerably smaller objects may be seen under chest roentgenoscopic conditions than at abdominal roentgenoscopy. Also, for lateral abdominal work, the object size must be relatively large before it can be seen. Indeed, objects of 10 per cent contrast must be 10 mm. in diameter before they may be detected.

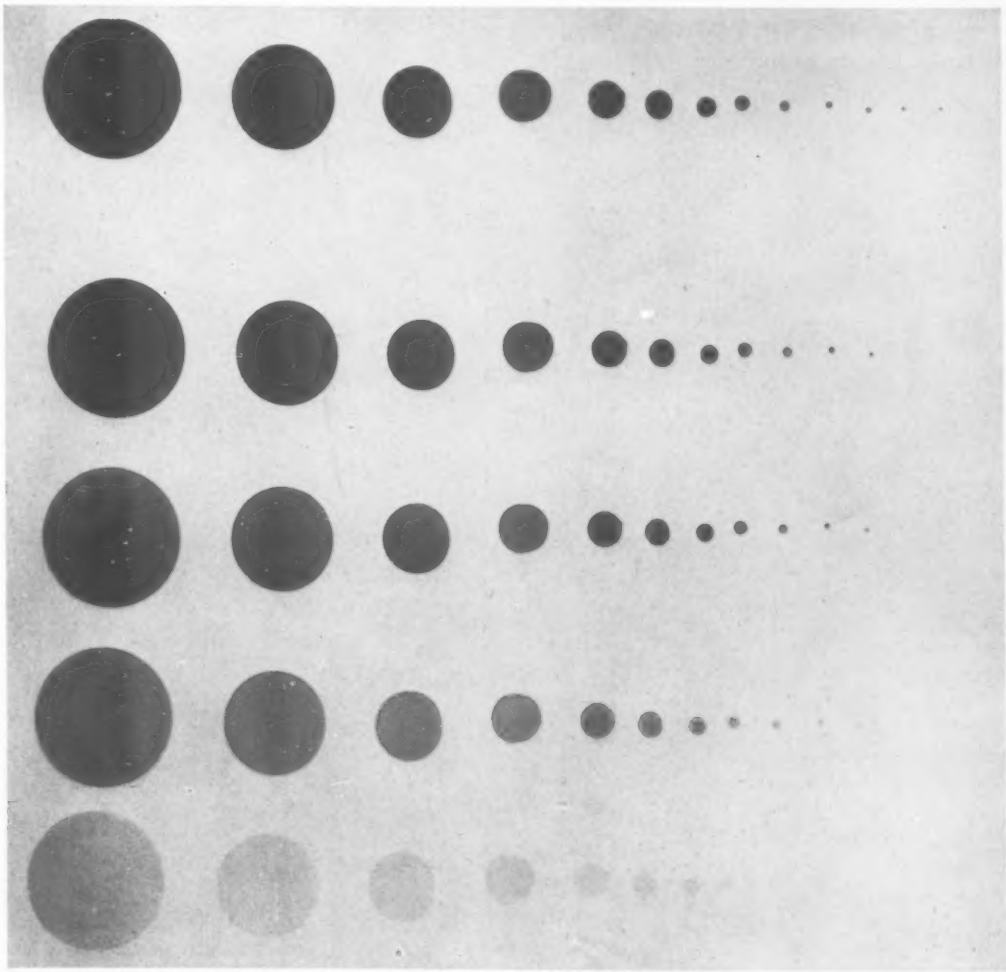

FIG. 6. Portion of the test pattern used in the experimental determination of visual performance at roentgenoscopic levels of scene luminance.

At this point, one may reasonably inquire about the correlation that exists between the purely theoretical calculations that have just been made for the visual performance of roentgenoscopy and experimental measurements of this same performance. Obviously, if the correlation is poor, there is little justification in carrying these discussions further in an attempt to

predict the visual responses which may occur at roentgenoscopy when the eye is aided by screen intensification. Reliable data giving the size and contrast of objects that are just perceptible under various roentgenoscopic conditions have not heretofore been published. Accordingly, an experimental investigation by which such data could be collected was established. A lead plate in which were perforated holes varying in size from 1 mm. to 22.4 mm. in diameter was prepared and roentgenographed several times on a film 8 by 10 inches in size. The several exposures were varied through a sufficient range that the images of the holes appearing on the film presented contrasts with their surrounding fields ranging from 2.5 per cent to 94 per cent. The film (Fig. 6) was then mounted on the face of a Patterson type B-2 screen and after the edges of the screen beyond the film were masked off, the screen-film combination was mounted in a typical roentgenoscopic unit. A roentgen beam was then projected on the screen and adjusted to give yields of 1.2×10^{9}, 1.3×10^{8}, and 1.7×10^{7} light photons per second per square millimeter of screen surface. As previously pointed out these yields are closely equivalent to those occurring at chest, anteroposterior abdominal and lateral abdominal roentgenoscopy. Six observers, free of optical defects, were then taken into the experimental room and allowed to become dark adapted for a period of one hour. Each observer was then placed in turn before the roentgenoscope at a viewing distance of 200 mm., and observations of the smallest perceptible object for each of the several contrast levels were made. Observations were first made at the screen luminance of lowest intensity, then the intermediate intensity, and finally the highest intensity. The averaged data for the six observers are shown graphically in Figure 7, where object size is plotted against object contrast. The solid lines represent the experimental observations while the dotted lines represent the calculated values illustrated in Figure 5.

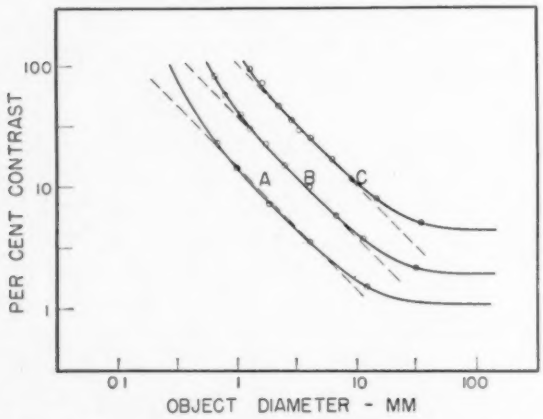

FIG. 7. Experimental visual performance data obtained under roentgenoscopic conditions equivalent to *A*, roentgenoscopy of the chest; *B*, anteroposterior roentgenoscopy of the abdomen and *C*, lateral abdominal roentgenoscopy. Screen: Patterson type B-2; viewing distance: 200 mm.; tube voltage: 70 kvp.; tube current: 5 ma.; tube-screen distance: 27 inches.

If the agreement between experimental and calculated data seems striking, it must be remembered that the calculated data were obtained by applying to equation (19) parameter values that were determined under experimental conditions not dissimilar to those existing during our study. Hence the agreement is no more than might have been expected. In addition to this, however, we believe it is quite significant that the experimental curves, over major portions, exhibit the same slope as the calculated curves. This fact seems to justify considerable confidence in the fluctuation theory of vision for there is no other apparent explanation of the phenomenon.

It will be observed that when the size of an object is small or its contrast low, deviations from the calculated curves appear in the experimental data. The deviations from the calculated values for small objects may be explained by the presence of light dispersion within the eye. This dispersion reduces the contrast and border sharpness of the retinal images and accordingly makes the perseption of those images abnormally difficult. The effect is logically greatest when object size is small.

At low contrast levels, the deviations from the calculated curves may be traced

to another phenomenon of optical physiology. When an object under observation is small it is appreciated in its entirety by a single fixation of the eye. However, when the object becomes larger, eventually a size is reached beyond which the eye must scan the object with multiple points of fixation if complete comprehension is to be obtained. That is to say, the object eventually becomes greater than the eye's maximum field of appreciation. Now when an object is of such size that it covers more than the maximum field of appreciation, it is reasonable to expect that the eye's contrast discrimination will be no greater than that produced by an object of a size just sufficient to cover this maximum field. Therefore, as one progresses to larger object sizes, the experimental curves of visual performance may be expected to deviate from calculated values based on fluctuation theory. It is evident, therefore, that the deviations from fluctuation theory which occur in the experimental curves of Figure 7 in no way invalidate this theory. Instead, they are manifestations of phenomena which under certain circumstances impose additional limitations on visual performance. Thus there appears to be little doubt that visual performance may be reasonably predicted from fluctuation theory and that we are justified in proceeding now to a discussion of the visual performance that might be obtained with an ideal screen intensifying device.

IV. IDEAL SCREEN INTENSIFICATION SYSTEM

It is clear from fluctuation theory that one's visual performance at roentgenoscopy can be improved only by increasing the number of photons which reach and stimulate the eye each second from the fluorescent screen. Now such an increase could be achieved by raising the voltage and current of the roentgen tube or by decreasing the tube-screen distance of the fluoroscope. These procedures, however, are open to serious criticism since they expose the patient and roentgenologist alike to the hazards of excessive radiation. Another method of obtaining an increased number of photons for the retina is the application of some sort of screen intensifying device to the roentgenoscopic system.

Although several methods of screen intensification have been proposed in recent years, none has been developed sufficiently far to permit intensive clinical trial. Therefore, it is not possible at this time to evaluate the improvement in visual perception that may be obtained with any of these methods. It is possible, however, from fluctuation theory, to determine the performance characteristics of an ideal screen intensifier. Although such a device is entirely hypothetical, it does represent the ultimate objective toward which all work in the field of screen intensification is being directed and its performance, accordingly, represents the maximum improvement in visual resolution and contrast discrimination that may be expected from screen intensifying instruments. The consideration of such a device will therefore give us at once a clear picture of what eventually may be gained by brightening the roentgenoscopic screen.

An ideal screen intensification system may be defined as one which absorbs completely all of the incident roentgen radiation falling upon it, one which does not introduce into the roentgenoscopic process any significant fluctuation and one in which the gain is controllable through a limitless range of intensification. For purposes of discussion, let us apply such an ideal screen intensifier to a roentgenoscope in which the roentgen tube is operating at 70 kvp. and 5 milliamperes, in which the roentgen beam is filtered by 1 mm. of aluminum and in which the distance between the roentgen tube and the pick-up surface of the screen intensifier is 27 inches. We have seen in preceding sections that, under these conditions, the roentgen tube projects toward each square millimeter of the intensifier's pick-up surface 5×10^6 roentgen-ray photons per second. We have also seen that when a patient is placed in the roentgen beam this number drops to 5×10^5 photons per second for roentgenoscopy of the chest, 5×10^4 photons per second for roentgenos-

copy of the abdomen in the anteroposterior projection and 5×10^3 photons per second for roentgenoscopy of the abdomen in the lateral projection. When these photons fall on the pick-up surface of the intensifying instrument, their energy is utilized in the production of light photons which form the visible image, perceived by the observer.

Now when the gain control of the ideal screen intensifier is adjusted to produce a viewing-screen brightness equivalent to that of a Patterson type B-2 screen at simple fluoroscopy and when the viewing distance of the observer is 200 mm., the visual performance of the observer will simply be that given by the previous curves for simple fluoroscopy (see Fig. 7). The series of events which transpire during the use of the ideal intensifier under these circumstances is shown graphically in Figure 8*A* for each of the three types of fluoroscopy. It will be observed that these events are similar to those occurring in conventional roentgenoscopy and depicted in Figure 4 except for the total roentgen-ray absorption which we have assumed for the ideal screen intensifier.

Now if the gain control of the ideal intensifier is slowly advanced so that the amount of intensification provided by the device becomes greater than that shown in Figure 8*A*, the number of light photons stimulating the observer's retinae will eventually become equal to the number of roentgen-ray photons absorbed by the intensifier's pick-up surface (Fig. 8*B*). During this phase of increasing screen luminance, it will be evident from equation (19) that there will occur a simultaneous improvement in the observer's visual performance.

Let us now continue to advance the intensifier's gain control so that the number of photons stimulating the retina rises above the number absorbed by the intensifier's pick-up surface (Fig. 8*C*). During this phase of increasing screen luminance, no improvement in visual performance can occur for, under these conditions, the stage of the roentgenoscopic process at which light photons stimulate the retina is no longer the stage of lowest intensity. Instead

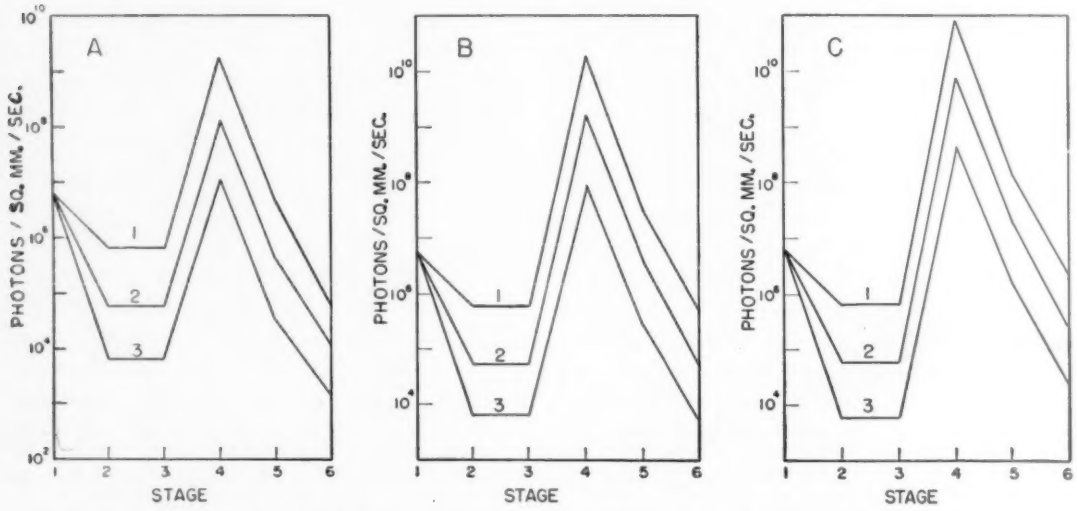

Fig. 8. Graphic representation of the photon densities that might exist during roentgenoscopy when aided by an ideal screen intensifier. *A*: condition where the gain control of the intensifier is retarded to the level at which screen luminance is equivalent to that occurring at simple roentgenoscopy; *B*: condition where the gain control is advanced to the level at which the screen luminance is such that the number of light photons stimulating the retina from each square millimeter of the intensifier's surface is equal to the number of roentgen-ray photons absorbed by the intensifier; *C:* condition where the gain control is advanced beyond *B*. Stages 1 to 6 are of the same significance as those shown in Figure 4. Curve 1: chest roentgenoscopy; curve 2: anteroposterior abdominal roentgenoscopy; curve 3: lateral abdominal roentgenoscopy.

the stage of roentgen-ray photon absorption by the intensifier's pick-up surface constitutes the stage of lowest intensity and accordingly the value of g, the amplification factor of the roentgenoscopic process between the stage of lowest intensity and the final stage, in equation (19), is no longer unity. Indeed it quite evident that after the number of photons stimulating the retina exceeds the number of roentgen-ray photons absorbed by the ideal intensifier, a rise in the number of retinal photons is accompanied by a proportionate rise in the amplification factor, g, of the system. Therefore, the effect of increases of n_0 in equation (19) is cancelled by the simultaneously occurring increases in g and no change in visual resolution or contrast discrimination occurs.

This situation has immediate and far-reaching implications for the roentgenologist for it imposes upon even the most perfect screen intensification system well-defined limits to the improvement in visual performance that may be obtained during roentgenoscopy. Contrary to widespread impression, a limitless improvement in visual acuity and contrast discrimination cannot be obtained just by increasing the luminance of the roentgenoscopic screen. Indeed, it will be evident from a perusal of Figures 4 and 8 that no improvement in visual performance will be obtained by screen intensification beyond that produced by an ideal intensifier having a luminance of 30 to 50 times greater than that of a Patterson type B-2 screen. An increase of brightness beyond this range will only decrease the need for dark adaptation; it will not increase the acuity and contrast discrimination of the observer.

The ultimate limits of visual performance that may be obtained by screen intensification may be easily determined for roentgenoscopy of the chest, of the abdomen in the anteroposterior projection and of the abdomen in the lateral projection by applying in equation (19) values of 5×10^5, 5×10^4, and 5×10^3 respectively for n_0, a value of 1.0 for g, and of course values of 5.0 for k and 0.2 for t. Calculations derived from these values are shown by the curves in Figure 9. In all probability these curves are valid only in their mid-portions since it is likely that the limits of visual performance, when objects of small size or low contrast are viewed, are contracted beyond the limits set by fluctuation theory. At least such was the case for conventional roentgenoscopy (see Fig. 7) and there is no reason to believe that screen intensification will alter this situation. Therefore one may expect that visual performance will be somewhat poorer than that indicated in Figure 9.

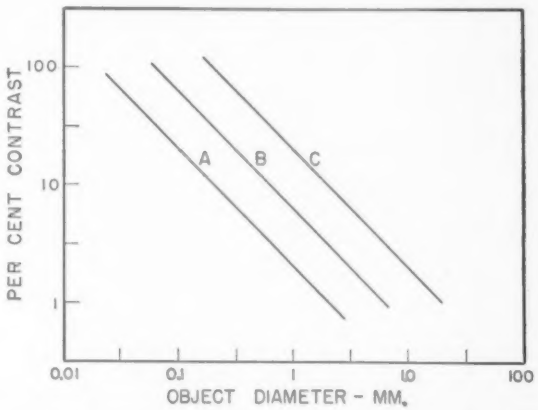

Fig. 9. Theoretical visual performance curves that might be obtained with an ideal screen intensification system. The designations A, B, and C refer to the same conditions as those specified in Figure 4.

V. Visual Performance at Roentgenography

Let us now consider the visual performance of an individual observing a roentgenographic film. In conventional practice, roentgen-ray photons are projected through the anatomical structure under examination and subsequently are allowed to fall on a pair of calcium tungstate intensifying screens. The fluorescence from these screens exposes the roentgenographic film and produces in it an image which appears after the film is processed. The image is then viewed by placing the film on an illuminator usually having a luminance of about 100 foot-lamberts. Now Coltman *et al.*[4] have shown that for each roentgen-ray photon absorbed by calcium tungstate screens, approximately 500 useful light

photons are produced. Also, estimates of the quantum efficiency of photographic and roentgenographic emulsions based on experimental evidence yield values in the neighborhood of 1 per cent. Therefore, approximately 5 developable photographic grains are produced in the roentgenographic film for each absorbed roentgen-ray photon, and accordingly the stage at which maximum fluctuation is introduced into the process is the stage at which roentgen-ray photons are absorbed by the intensifying screens. Additional fluctuations also occur during the viewing of the film, but by using relatively high levels of illumination (e.g. 100 foot-lamberts) and short viewing distances (e.g. 200 mm.) these fluctuations may be maintained at comparatively insignificant levels.

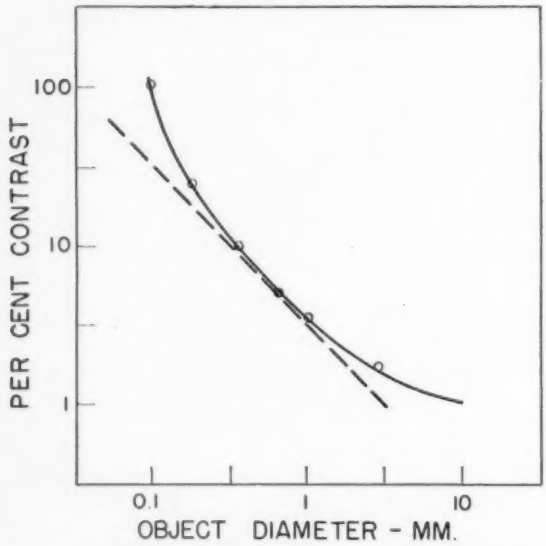

FIG. 10. Visual performance curve of Eastman blue brand film exposed with Patterson par speed intensifying screens.

Recent experimental work[9] indicates that a typical roentgenographic film-screen combination (Eastman blue brand film and Patterson par speed intensifying screens) when exposed with a 70 kvp. roentgen beam having a half-value layer of 4 mm. of aluminum requires an exposure of 0.001 roentgen to produce a film density of 1.0, the average density of a correctly exposed roentgenogram. From equation (20) such an exposure corresponds to 6×10^4 roentgen-ray photons per square millimeter. Now the absorption of Patterson par speed screens with such a beam is approximately 70 per cent. Therefore, 4×10^4 roentgen-ray photons per square millimeter are responsible for the production of the roentgenographic image. When such a value is substituted for the product of $n_0 t/g$ together with the usual value of 5.0 for k in equation (19) the visual performance of a typical roentgenographic film as limited by fluctuation theory may be easily shown to be that illustratetd by the dotted curve in Figure 10.

Data on the actual performance of Eastman blue brand film exposed with Patterson par speed screens are not available in the literature for comparison with this theoretical curve. Therefore, such data were obtained experimentally in the laboratory by exposing the film-screen combination with a lead plate having perforations ranging in size from 0.1 mm. to 10 mm. The exposure contrast was varied through a range of 1 per cent to 100 per cent by giving the combination pre-exposures sufficient to yield a total exposure in each case of 0.001 roentgen. The exposures were produced by a 70 kvp. roentgen beam having a half-value layer of 4 mm. of aluminum. The film was then processed and viewed at a distance of 200 mm. on an illuminator having a luminance of 100 foot-lamberts. The results of the test are shown by the solid curve in Figure 10. The close correlation of experiment and theory is indeed striking and gives additional support, if such support is needed, to the importance of fluctuation theory in visual perception. The correlation demonstrated in Figure 10 is particularly significant because it illustrates the correctness of fluctuation theory in a condition where the amount of fluctuation is determined by the number of roentgen-ray photons absorbed by the photosensitive medium. The curves of visual performance for the ideal screen intensifier in Figure 9 were based on a similar condition, and although an ideal screen intensifier is not available for test, the agreement between theory and experiment in Figure 10 indicates that the curves are probably correct.

As was the case in roentgenoscopic vision, there is some deviation between the experimental and predicted curves for objects of small size and low contrast in roentgenography. Such deviations, however, may be reasonably explained on the same basis as that given previously in the section on simple roentgenoscopy and certainly do not in any way invalidate fluctuation theory as a limiting factor in roentgenographic perception.

The experimental curve shown in Figure 10 is applicable with very minor modifications to roentgenography of any anatomical structure since approximately the same number of absorbed roentgens are needed to produce a film of an acceptable density throughout the range of anatomical thickness encountered in medical roengtenography. It will be observed that the roentgenograpic film has the great advantage over roentgenoscopy in that as the brightness of the intensifying screens diminishes with increasing anatomical thickness, the exposure time, or more precisely the storage time of the film, increases reciprocally to maintain the observer's visual performance at a high level. In roentgenoscopy, the storage time of the recording medium is simply that of the observer's eyes. This time, as we have seen, is independent of screen illumination and accordingly visual performance deteriorates when heavy structures are roentgenoscoped. In Figure 11, the visual performance curves of simple roentgenoscopy, of roentgenoscopy aided by an ideal screen intensifier and of roentgenography are plotted for three different conditions. Figure 11*A* applies to the examination of the chest, Figure 11*B* to the examination of the abdomen in the anteroposterior projection, and Figure 11*C* to the examination of the abdomen in the lateral projection. It will be seen that only in the examination of the chest does the performance of the ideal screen intensifier approach that of roentgenographic film. In the examination of the abdomen its performance begins to fall short of that of film and this is particularly so when the structures under examination are heavy. How-

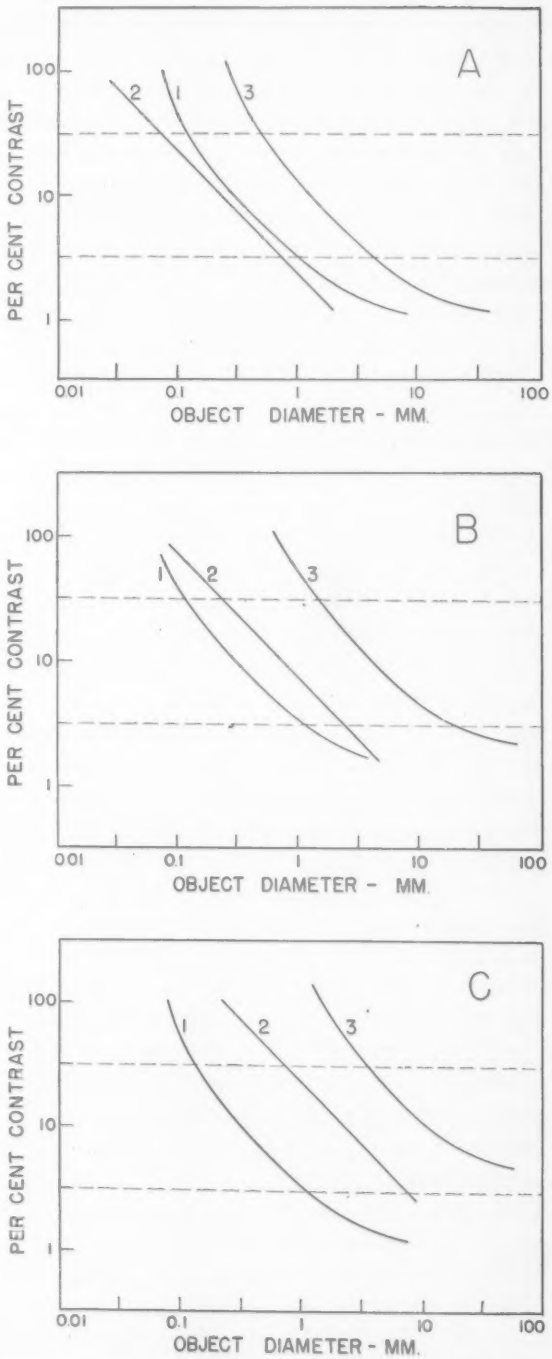

Fig. 11. Visual performance data showing the comparative merits of (1) roentgenography; (2) an ideal screen intensifier; and (3) simple roentgenoscopy. *A* refers to conditions equivalent to those occurring during the examination of the chest; *B* to conditions equivalent to those occurring during the examination of the abdomen in the anteroposterior projection; and *C* to conditions equivalent to those occurring during the examination of the abdomen in the lateral projection.

ever, even though these benefits to be derived from screen intensifications are more limited than many had anticipated, they are nevertheless sufficiently impressive to warrant enthusiastic anticipation of practical intensifying devices. Of course, it is unlikely that such instruments when they become available will approach the ideal curves shown in Figure 11. For several years there doubtless will be an evolutionary period during which the available screen intensifiers will fall short of the ideal intensifier. However, there appears to be no reason why screen intensifying devices providing very real benefits should not eventually be obtainable.

This, then, is the outlook for screen intensification. Perhaps it is not as impressive as many had hoped for. It must be emphasized again, however, that the fundamental limitation on visual performance during screen intensification is the number of roentgen-ray photons absorbed by the intensifying device and we are indeed fortunate that reasonably large improvements are attainable within the limits of roentgen exposure imposed by sound clinical practice. It should also be pointed out that although the maximum improvement in visual acuity and contrast discrimination that may be obtained with an ideal screen intensifier will occur at gains of 30 to 50 times over the Patterson type B-2 screen, much higher gains will probably be desirable in practical instruments for several reasons. Gains of 500 to 1,000 times above the B-2 screen are needed if dark adaptation is to be avoided. Such gains also may be necessary if the image quality of the instrument is to approach that of the ideal screen intensifier. Hence, the need of energetic investigation which will lead to the development of high gain screen intensifiers cannot be questioned. It appears, however, that the benefits derived from such intensification will be principally concerned with the elimination of dark adaptation rather than with enormous gains in the observer's visual acuity.

VI. SUMMARY

The benefits to be derived from screen intensification systems are predicted by calculating the visual performance of an ideal screen intensifier from statistical fluctuation theory. It is shown that for roentgenoscopy of the chest and of other structures of comparable radiolucency one may ultimately expect the visual performance of screen intensifiers to approach that of roentgenographic film. Roentgenoscopy of the abdomen and similar structures, however, will not likely be as satisfactory due to limitations imposed by the amount of roentgen exposure which may be safely tolerated by the patient and roentgenologist.*

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* For discussion, see page 637.

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CLINICAL POTENTIALITIES OF SCREEN INTENSIFYING SYSTEMS*†

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IT IS perhaps idle and even foolhardy to discuss the clinical potentialities of screen intensification systems when a practical device providing an appreciable increase in screen brightness is not yet available. However, the information given in earlier papers this afternoon by Sturm and Morgan and by Coltman** makes it possible to anticipate, in part at least, the course which roentgenology may take when bright fluoroscopic screens can be obtained.

At the outset, it is clear that the benefits to be derived from screen intensification are sufficiently great to insure a much wider use of roentgenoscopy than at present. Not only may the roentgenologist be able to see more clearly than in the past, but with the gains in screen brightness of five hundred times expected with the Coltman image tube, screen luminance will be sufficiently great to be well within the range of cone vision even when the thickest anatomical structure is examined. Thus long periods of dark adaptation before roentgenoscopy will no longer be necessary and the fluoroscope will attain a degree of convenience heretofore unknown.

The wider use of roentgenoscopy will doubtless have a number of significant ramifications. First, the method by which roentgenology is practiced will undergo important and beneficial changes. Henceforth the roentgenologist will be brought into closer and closer contact with his patients under conditions more conducive to a satisfactory patient-physician relationship. The patient's apprehensiveness occasioned by the totally darkened roentgenoscopic room will be gone; the technical nature of the examination will be subordinated, and the professional role of the roentgenologist will become increasingly apparent to the patient. These are certainly changes that will be welcomed by everyone, for their medico-social implications are obvious.

The wider use of roentgenoscopy among roentgenologists may be expected to extend to those physicians who are not fully qualified in the use of roentgen methods. Although this is an undesirable situation, it is one that presents one advantage. The use of roentgenologic methods in the diagnosis of disease will be increasingly emphasized and the need of competent roentgenologic assistance will ultimately be apparent to all conscientious physicians. It does not appear that the roentgenologist has anything to fear from the increased ease with which his tools may be used following the introduction of screen intensification methods. On the contrary, this evolution may be expected to lead only to a more prominent place for the roentgenologist in the practice of medicine.

The increased application of roentgenoscopy will not be without its technical dangers, for the roentgenologist will be exposed to quantities of radiation considerably in excess of those currently encountered unless more rigid protective measures than those now followed are employed. Fortunately, the greater visibility of the screen will tend to reduce the exposure of patient and physician. Furthermore, as inferred by Sturm and Morgan, the roentgen beam may be reduced in intensity, if desired, by a factor of 30 to 50 times when an ideal screen intensifier is used without reducing the clarity of per-

** See Coltman, J. W. Fluoroscopic image brightening by electronic means. *Radiology*, 1948, *51*, 359–367.

* From the Department of Radiology, The Johns Hopkins Hospital and University. Presented at the Forty-ninth Annual Meeting, American Roentgen Ray Society, Chicago, Ill., Sept. 14–17, 1948.
† The work presented in this paper was supported by a grant from the National Cancer Institute, the National Institute of Health, United States Public Health Service.

ception below that now obtainable at roentgenoscopy. However, if the benefits of increased perception provided by screen intensification are to be achieved, it is fairly evident that such reductions in beam intensity should not as a rule be made, but instead efforts should be directed toward improvements in the protection afforded the roentgenologist.

The clinical benefits of screen intensification are of course enormous and appear to be greatest in the examinations of the chest and of the extremities where the visibility of the screen may be expected to approach that of roentgenographic film. However, unlike film which restricts the roentgenologist to, at most, a few glimpses of the structure of interest, the intensified fluoroscopic screen will provide an almost limitless scrutiny of the patient. The detection of pathological anatomy will thereby be obviously facilitated.

Of even greater importance than the ease with which pathological changes may be elicited, will be the facility with which normal and abnormal physiological states may be studied. For example, the physiological phases of orthopedics will be amenable to a thoroughness of investigation heretofore unattainable by other methods. Certainly the field of roentgen physiology will offer an immediate challenge to countless roentgenologists in the study of many anatomical systems just as soon as practical intensifying instruments are available.

Screen intensification methods will probably accentuate the place of photofluorography as a diagnostic aid. It is anticipated that physicians will always wish to have pertinent film evidence of abnormal changes within a patient for comparison at subsequent examinations. In many respects, photofluorography offers an ideal solution to this problem. With the increased screen brightness provided by intensification systems, photofluorographic apparatus using lenses of smaller aberration and films of finer grain may be expected to produce photofluorograms having a clarity equivalent to that of standard roentgenograms. If such is the case, a patient's permanent film record may be obtained more quickly and easily than is possible with present-day roentgenographic equipment. The advantages of the small film in its filing will also be very worth while.

The barriers to practical roentgen cinematography will largely be removed by screen intensification systems, for the now excessive exposures of patient and equipment should then be no longer necessary. The study of cardiac physiology by means of opaque material, and the study of the dynamics of the musculo-osseous system may be expected to be greatly benefited by these developments.

It is of course impossible to anticipate at this time more than a few of the results that will occur when instruments for intensifying the fluoroscopic screen become available. One thing is sure, however, and that is that roentgenology will assume a position of importance in the practice of medicine unequalled heretofore. A few problems, particularly in the field of protective technology, will be introduced by this new development. However, the experimental, clinical, and social benefits are so enormous that they far outweigh these minor disadvantages. Only after the next few years will we begin to appreciate the exact significance of this new adjunct to roentgen diagnosis. From the foregoing, it must not be inferred that screen intensification systems are likely to revolutionize the practice of roentgenology. Such a development is far from probable. Instead it appears more reasonable to expect that the availability of screen intensifiers will begin a systematic evolution of roentgenologic practice which will extend over a period of many years. At first, the new screen intensifiers may fall far short of the ideal as far as clarity of reproduction is concerned. The first instruments also may not be as convenient in use as desired. For example, the Coltman image tube will require the roentgenologist to observe the brightened fluoroscopic screen through a telescopic eyepiece; such a viewing method will ob-

viously be clumsy and unsatisfactory to most roentgenologists. However, as time progresses, new developments will bring practical screen intensification systems even closer to the ideal until ultimately intensifying devices, simple in use, clear in performance, and, it is hoped, inexpensive in price will be generally available to the profession.

Johns Hopkins Hospital
Baltimore 5, Md.

DISCUSSION OF SYMPOSIUM ON THE INTENSIFICATION OF THE ROENTGENOSCOPIC SCREEN*

Dr. Paul C. Hodges, Chicago, Ill. It was fitting and right that Dr. Chamberlain's paper† should have been the first one on this program today because in this as in so many other phases of roentgenology, Dr. Chamberlain's ideas and work have started a whole train of events. Dr. Chamberlain was a prime mover in pelvimetry and cardiology and now has stimulated our interest in amplification of fluoroscopic images.

It was Chamberlain's ideas that led Dr. Morgan to be interested in image amplification when he was with us at the University of Chicago and, as you have heard here today, that interest has been continued and intensified now that he has his own department at Johns Hopkins University.

I shall make my own discussion of these papers as brief as possible so that time may be made available for Professor Moon. Moon had been invited to present a paper in this symposium but at the time of the invitation it seemed unlikely that his work would be far enough advanced to warrant a report. He requested, therefore, that he be allowed merely to discuss these other papers. As a matter of fact, his work has progressed more rapidly than we anticipated and it has seemed to us that the purpose behind this symposium will best be served by having him devote his time to telling you of the progress that he has made in the particular form of amplification that he published in the June, 1948 number of this Journal.

As to the paper by Drs. Sturm and Morgan, I know from long experience that a great many of Dr. Morgan's hunches do turn out to be correct but he would be the first to tell you that sometimes they are wrong. I hope that this time he and Dr. Sturm are a little too conservative and picture the scene a little darker than it really is.

In regard to Dr. Coltman's paper, the simplicity of his design is a desirable feature and one that justifies the hope of early and relatively inexpensive adaptation to clinical use but, as he points out, the amount of amplification presently available is not very great. I am sure he will agree that the search must go on until we have attained a maximum of brilliance and resolving power.

Perhaps the optimum system will prove to be a modification of the tube Dr. Coltman is using today or possibly it will be something that today is totally unthought of. If the best image amplification system that can be conceived by man proves to be, in its experimental phase, as large as a railroad car, obviously it will have no clinical use in that stage of development. Almost surely, however, if such a system proves to be desirable in all respects other than size, engineers will find some means to refine it and reduce its bulk until it can be used clinically.

As to the smallness of the size of the fluoroscopic screen, I agree completely with Dr. Coltman that this is not a defect. For many years we have done all of our gastric fluoroscopy with a $4\frac{1}{2}$ by 7 inch fluoroscopic screen and find that size adequate.

Dr. Robert J. Moon, Chicago, Ill. When Dr. Paul Hodges presented the problem of the amplification of the fluoroscopic image to me about a year ago in a casual conversation, it was just one of those interesting discussions at the time. However, it was well to have this information in mind; for subsequent developments that occurred last February in some work which we were doing in our Institute of Radiobiology and Biophysics appeared to be immediately applicable to the amplification of the roentgenoscopic image. A scheme for the solution of Dr. Hodges' problem was drawn up and discussed with him, the result of which was that he urged this line of approach be investigated as to its possibilities. Preliminary measurements showed the feasibility of the idea. This particular method

* This work has been supported in part by grants from the Wallace O. and Clara A. Abbott Fund of the University of Chicago, the Office of Naval Research, the United States Public Health Service, and the Industrial Sponsors of the Institute.

† Dr. Chamberlain's paper is not available for publication at this time.

was described in a communication which appeared in the June, 1948, issue of the American Journal of Roentgenology and Radium Therapy. The general scheme of the method is shown in Figure 1 of that paper.

About three months ago, the above paper was discussed with Dr. Szegho, Dr. Polanyi and Dr. Marcy of the Rauland Corporation, and these gentlemen were all very much interested in the general nature of the problem and specifically in the adaptation of one of their projection kinescopes as a scanning roentgen tube for the amplification of the fluoroscopic image. Two such scanning roentgen tubes are now in operating condition. We took our first photograph with such a tube last night, although we had been observing some images with the tube in the past few weeks.

The spot where the cathode particle strikes the target in the roentgen tube moves back and forth across the target and after each transit it takes another path across which is below the preceding path. This is continued until the bottom edge of the target is reached at which time the spot returns to the upper edge of the target and repeats its trip across the target as before. The same procedure is repeated many times a second. This of course is just the way the spot scans the face of a picture tube in a television receiver. The roentgen rays which are generated at the spot where the cathode particles strike the anode are permitted to escape only through a tiny pinhole which is essentially part of the wall of a lead box which encloses this scanning roentgen tube. Thus a tiny search light of roentgen rays is generated which is scanning back and forth with its virtual origin at the pinhole. Thus, after a patient is placed in this roentgen-ray field and the roentgen rays are allowed to fall on a fluorescent screen, which is placed opposite the patient from the pinhole, a roentgenoscopic image would be seen just like that with which you are familiar. There is one essential difference, however; if the screen were observed for a very short instant, say a microsecond, and if a fast fluorescent screen were employed, then only a spot would be seen which would correspond to the position of the roentgen rays at that instant. The fluorescent light pulses from each particular spot are picked up by the photocell and the photocurrent amplified so that it can modulate the intensity of the cathode-ray stream of a kinescope in order that the picture may be reconstructed on a picture tube just as it is done in television. In the particular experiments which are referred to above, a photograph of a roentgen-ray shadow of an image of a small gear was taken with a 525 line picture (transits of the roentgen-ray beam per frame) with thirty frames per second. The photograph is not of the direct fluorescent screen but of the screen of the kinescope.

Many problems were encountered in the development of this system and the progress has been good towards their solution. One problem was that of finding suitable fluorescent screens which had an extremely short-time fluorescence. By short, I mean something less than a microsecond. It seemed that some of the screens which we had used in the laboratory would be very suitable and then, however, it turned out that most of these had a persistence in some cases as great as 60 microseconds and were not useful. A screen which Mr. Patterson and Mr. Reuter had sent to Dr. Hodges proved to be satisfactory. It is a screen of lead barium sulfate which has an extremely short persistence, and when used at ordinary room temperatures the persistence is imperceptible, though when cooled with liquid nitrogen, the persistence is of the order of 60 microseconds. However (since it is undesirable that it should be necessary to provide for the refrigeration of a fluorescent screen as was thought might be the case from some earlier experiments), this particular screen is most suitable because of the above two reasons for this particular application.

As mentioned above, the fluorescent spot of roentgen rays strikes the screen after passing through the object and a fraction of the fluorescent pulse of near visible light which is generated strikes the photocell. The pulse of photocell current is then amplified by means of a wide band amplifier. Thus amplification both as to picture size and intensity is accomplished electronically and the final image appears on the kinescope. Of course, more than one kinescope can be used for observation by several observers as well as one being reserved for the purpose of being photographed from time to time.

The picture to which I referred above required only about a tenth of a milliroentgen. The photograph is of the kinescope, and the exposure time is in the order of a second. The image seemed to have sufficient quality for television broadcast. Before I show a slide of this photograph I should like to use a slide which

pictures the first model of the scanning roentgen tube.

The first slide is a picture of one of an actual scanning roentgen tube (Fig. 1). On the left side of the tube is seen a small narrow neck which contains the electron gun. The electrons which are emitted from the gun are then accelerated by the electric field which is created by the potential difference between the gun and target

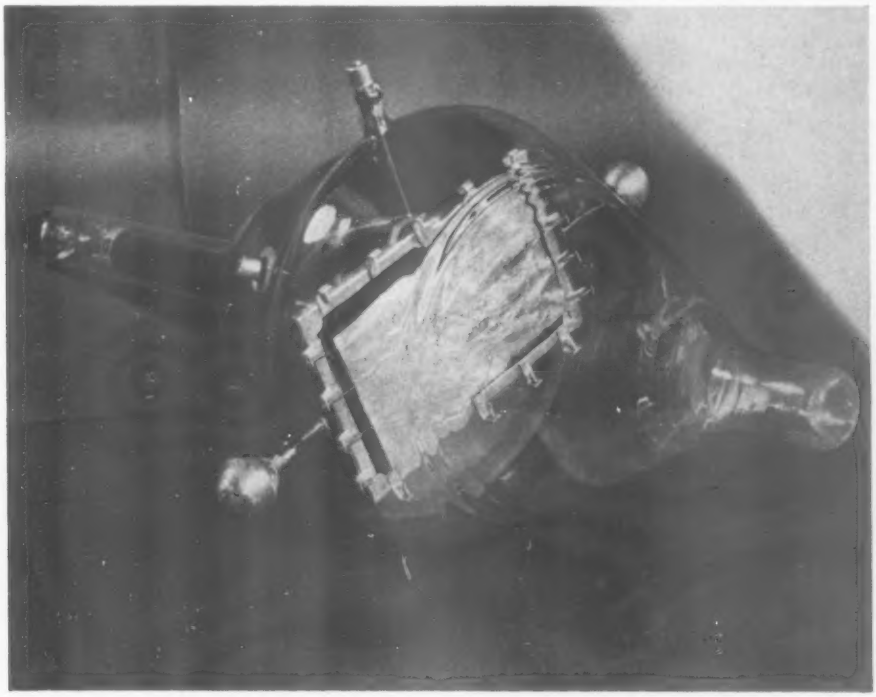

Fig. 1

of some 60,000 volts in this particular tube. The anode or target of this tube consists of a tantalum foil which is one-thousandth of an inch thick and approximately 4 by 7 inches. The foil is stretched by springs in order to keep it somewhat flat since it is operated at an incandescent temperature. This particular tube, as stated earlier, is an adaptation of a projection television kinescope and was made for us by one of the local television tube manufacturers, namely the Rauland Corporation. Essentially the fluorescent screen was cut off and the tantalum target put in its place. The tube was then extended as seen to the right of the picture, and finally terminated in a thin bubble window. The target appears to be somewhat distorted in the slide because of the wrinkles in the glass envelope. Since the roentgen rays are generated where the electrons strike the foil, they then have to pass through the foil in order to reach the window. This particular angle of the roentgen rays with respect to the electron beam is very near the minimum of roentgen ray intensity and in addition there is the further factor of attenuation of the roentgen radiation by the tantalum foil.

A small lead diaphragm which has an aperture of 0.45 mm. in diameter is placed in position to the right of the bubble window which is approximately 30 centimeters from the tantalum target. As stated earlier, this aperture is really part of the lead box which completely surrounds the scanning roentgen tube. To the right of this aperture, of course, objects are placed which are to be viewed roentgenoscopically. To the right of the object is placed a fast fluorescent screen. This detects the instantaneous number of roentgen-ray quanta which are able to pass through the patient in this narrow beam which has approximately a square millimeter area at the fluorescent screen and a time duration somewhat less than a microsecond. The photomultiplier tube responds to these fluorescent bursts of light and the signal is amplified by a wide band amplifier and the image recon-

structed on the fluorescent screen of a regular television picture tube.

The second slide is a roentgen-ray shadow image of a gear which was taken by means of this device (Fig. 2). This of course is a rather crude picture, first because it is a positive print of the actual photographic negative, and second, it is greatly magnified in size. However, since it is one of the first photographs of its kind it is indeed very gratifying to me personally that it is as good as it is. The size of the gear from tooth to tooth through the center is only 16 millimeters. There are some enlargements of the negatives here, which some of you might perhaps like to see after the meeting, in which the over-all magnification is fourteen times. The quantum nature of the roentgen rays is in evidence where the roentgen-ray attenuation is greatest such as in the shadows of the thick portions of the gear, where the picture appears to be constructed of tiny round spots instead of being uniform.

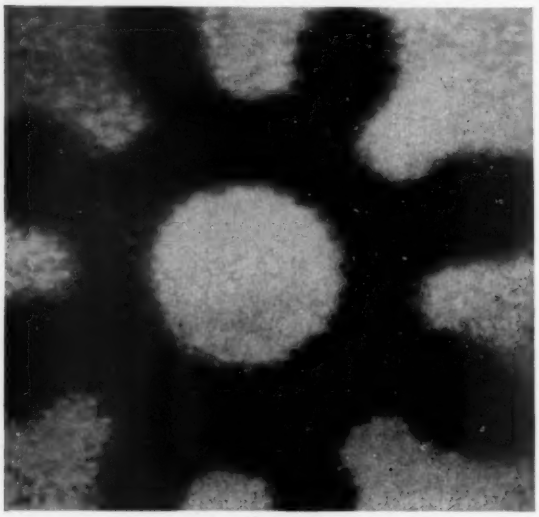

Fig. 2

Since the quanta are at various energies from 60 kilovolts to approximately one-fifth of that, the intensity of each individual fluorescent burst will vary approximately five to one with their most probable intensity corresponding, of course, to about one-half of the maximum electron energy, i.e. one-half of 60,000 electron volts in this case. This produces a snowstorm effect and would be visible on the regular fluorescent screen if it were not for the low light intensity. This effect can be nearly eliminated by means of a stripper amplifier which causes all quanta regardless of their energy (as long as they are above some set minimum value) to produce the same effect on the fluorescent screen of the picture tube. In other words, this stripper amplifier admits photocurrent pulses which are above a certain minimum value and clips all those photocurrent pulses which are above a certain maximum value. This maximum value is near that of the minimum value. Though the photocurrent pulses may vary five to one, this amplifier makes them appear identical or nearly so.

A roentgenoscopic shadow picture, which is constructed from the number of the roentgen-ray quanta which pass through the object at any one infinitesimal area, should be just as useful in constructing an image, if not more so, than the amount of roentgen-ray energy per infinitesimal area as is ordinarily used. The striper amplifier followed by a sufficiently wide band amplifier has shown considerable improvement in the picture and this approach has promise of greater improvement in the future. Some dots seen near the edge of the thick portion of the gear, which is in a region of nearly 100 per cent contrast with the surrounding region, are due to some extent to scattered roentgen rays and to a very small extent to persistence of fluorescence or delayed emission of fluorescent light pulses from the thick crystalline fluorescent screen.

The next slide shows another type of high energy kinescope, the design of which might perhaps be adopted to this problem (not illustrated here). It is a type of tube made by the Rauland Corporation and, as you see, looks very much like a pipe. The stem of the pipe contains the electron gun and the bowl of the pipe would contain a water-cooled anode or target for the roentgen tube. The roentgen rays would come from the surface of the tube and could be selected at an angle with respect to the primary electron beam which would yield a maximum intensity of the roentgen rays. The pinhole could be placed somewhat closer to the target. This particular design is an attempt to increase the number of roentgen-ray quanta which are available per picture element in order that the definition of the picture on the kinescope will not be limited because of statistical fluctuation resulting from too small a number of quanta per picture element.

As indicated previously, a rather small

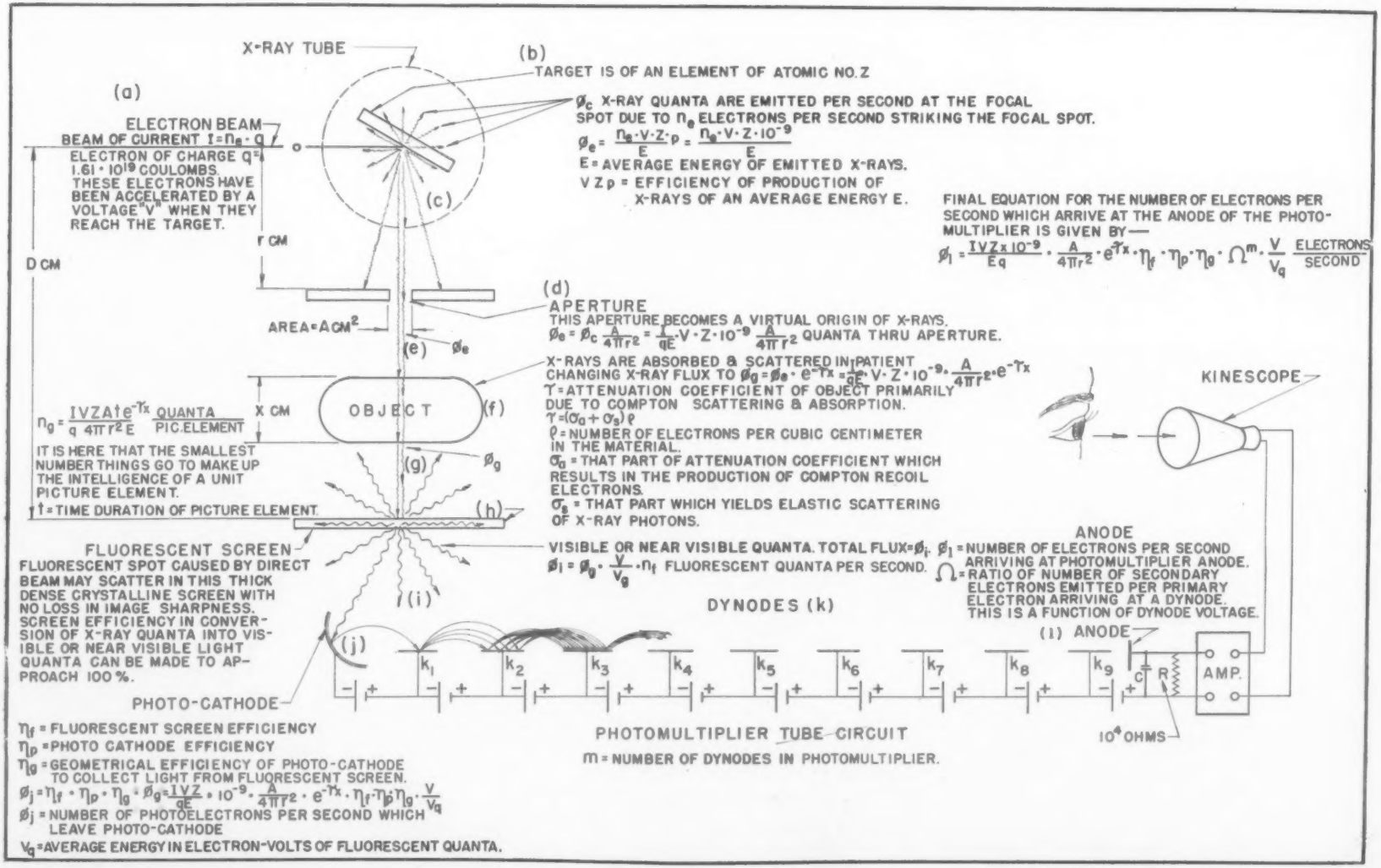
X-RAY TUBE
(a)
ELECTRON BEAM
BEAM OF CURRENT $I = n_e \cdot q$
ELECTRON OF CHARGE $q = 1.61 \cdot 10^{19}$ COULOMBS.
THESE ELECTRONS HAVE BEEN ACCELERATED BY A VOLTAGE "V" WHEN THEY REACH THE TARGET.
r CM
D CM
(b)
TARGET IS OF AN ELEMENT OF ATOMIC NO. Z
ϕ_c X-RAY QUANTA ARE EMITTED PER SECOND AT THE FOCAL SPOT DUE TO n_e ELECTRONS PER SECOND STRIKING THE FOCAL SPOT.
$\phi_e = \frac{n_e \cdot V \cdot Z \cdot p}{E} = \frac{n_e \cdot V \cdot Z \cdot 10^{-9}}{E}$
E = AVERAGE ENERGY OF EMITTED X-RAYS.
V Z p = EFFICIENCY OF PRODUCTION OF X-RAYS OF AN AVERAGE ENERGY E.
(c)
FINAL EQUATION FOR THE NUMBER OF ELECTRONS PER SECOND WHICH ARRIVE AT THE ANODE OF THE PHOTOMULTIPLIER IS GIVEN BY—
$\phi_1 = \frac{IVZ \times 10^{-9}}{Eq} \cdot \frac{A}{4\pi r^2} \cdot e^{-\tau x} \cdot \eta_f \cdot \eta_p \cdot \eta_g \cdot \Omega^m \cdot \frac{V}{V_q}$ ELECTRONS / SECOND
(d)
APERTURE
THIS APERTURE BECOMES A VIRTUAL ORIGIN OF X-RAYS.
$\phi_e = \phi_c \frac{A}{4\pi r^2} = \frac{I}{qE} \cdot V \cdot Z \cdot 10^{-9} \frac{A}{4\pi r^2}$ QUANTA THRU APERTURE.
AREA = A CM^2
(e) ϕ_e
X-RAYS ARE ABSORBED & SCATTERED IN PATIENT CHANGING X-RAY FLUX TO $\phi_g = \phi_e \cdot e^{-\tau x} = \frac{I}{qE} \cdot V \cdot Z \cdot 10^{-9} \cdot \frac{A}{4\pi r^2} \cdot e^{-\tau x}$
τ = ATTENUATION COEFFICIENT OF OBJECT PRIMARILY DUE TO COMPTON SCATTERING & ABSORPTION.
$\tau = (\sigma_0 + \sigma_s)\rho$
ρ = NUMBER OF ELECTRONS PER CUBIC CENTIMETER IN THE MATERIAL.
σ_0 = THAT PART OF ATTENUATION COEFFICIENT WHICH RESULTS IN THE PRODUCTION OF COMPTON RECOIL ELECTRONS.
σ_s = THAT PART WHICH YIELDS ELASTIC SCATTERING OF X-RAY PHOTONS.
X CM
OBJECT
(f)
$n_g = \frac{IVZAte^{-\tau x}}{q\,4\pi r^2 E}$ QUANTA / PIC. ELEMENT
IT IS HERE THAT THE SMALLEST NUMBER THINGS GO TO MAKE UP THE INTELLIGENCE OF A UNIT PICTURE ELEMENT.
(g) ϕ_g
t = TIME DURATION OF PICTURE ELEMENT.
(h)
FLUORESCENT SCREEN
FLUORESCENT SPOT CAUSED BY DIRECT BEAM MAY SCATTER IN THIS THICK DENSE CRYSTALLINE SCREEN WITH NO LOSS IN IMAGE SHARPNESS. SCREEN EFFICIENCY IN CONVERSION OF X-RAY QUANTA INTO VISIBLE OR NEAR VISIBLE LIGHT QUANTA CAN BE MADE TO APPROACH 100%.
VISIBLE OR NEAR VISIBLE QUANTA. TOTAL FLUX = ϕ_i.
$\phi_l = \phi_g \cdot \frac{V}{V_q} \cdot \eta_f$ FLUORESCENT QUANTA PER SECOND.
(i)
KINESCOPE
ANODE
ϕ_1 = NUMBER OF ELECTRONS PER SECOND ARRIVING AT PHOTOMULTIPLIER ANODE.
Ω = RATIO OF NUMBER OF SECONDARY ELECTRONS EMITTED PER PRIMARY ELECTRON ARRIVING AT A DYNODE. THIS IS A FUNCTION OF DYNODE VOLTAGE.
DYNODES (k)
(j)
k_1 k_2 k_3 k_4 k_5 k_6 k_7 k_8 k_9
(l) ANODE
C
R
AMP.
10^4 OHMS
PHOTO-CATHODE
PHOTOMULTIPLIER TUBE CIRCUIT
m = NUMBER OF DYNODES IN PHOTOMULTIPLIER.
η_f = FLUORESCENT SCREEN EFFICIENCY
η_p = PHOTO CATHODE EFFICIENCY
η_g = GEOMETRICAL EFFICIENCY OF PHOTO-CATHODE TO COLLECT LIGHT FROM FLUORESCENT SCREEN.
$\phi_j = \eta_f \cdot \eta_p \cdot \eta_g \cdot \phi_g = \frac{IVZ}{qE} \cdot 10^{-9} \cdot \frac{A}{4\pi r^2} \cdot e^{-\tau x} \cdot \eta_f \cdot \eta_p \eta_g \cdot \frac{V}{V_q}$
ϕ_j = NUMBER OF PHOTOELECTRONS PER SECOND WHICH LEAVE PHOTO-CATHODE
V_q = AVERAGE ENERGY IN ELECTRON-VOLTS OF FLUORESCENT QUANTA.

FIG. 3

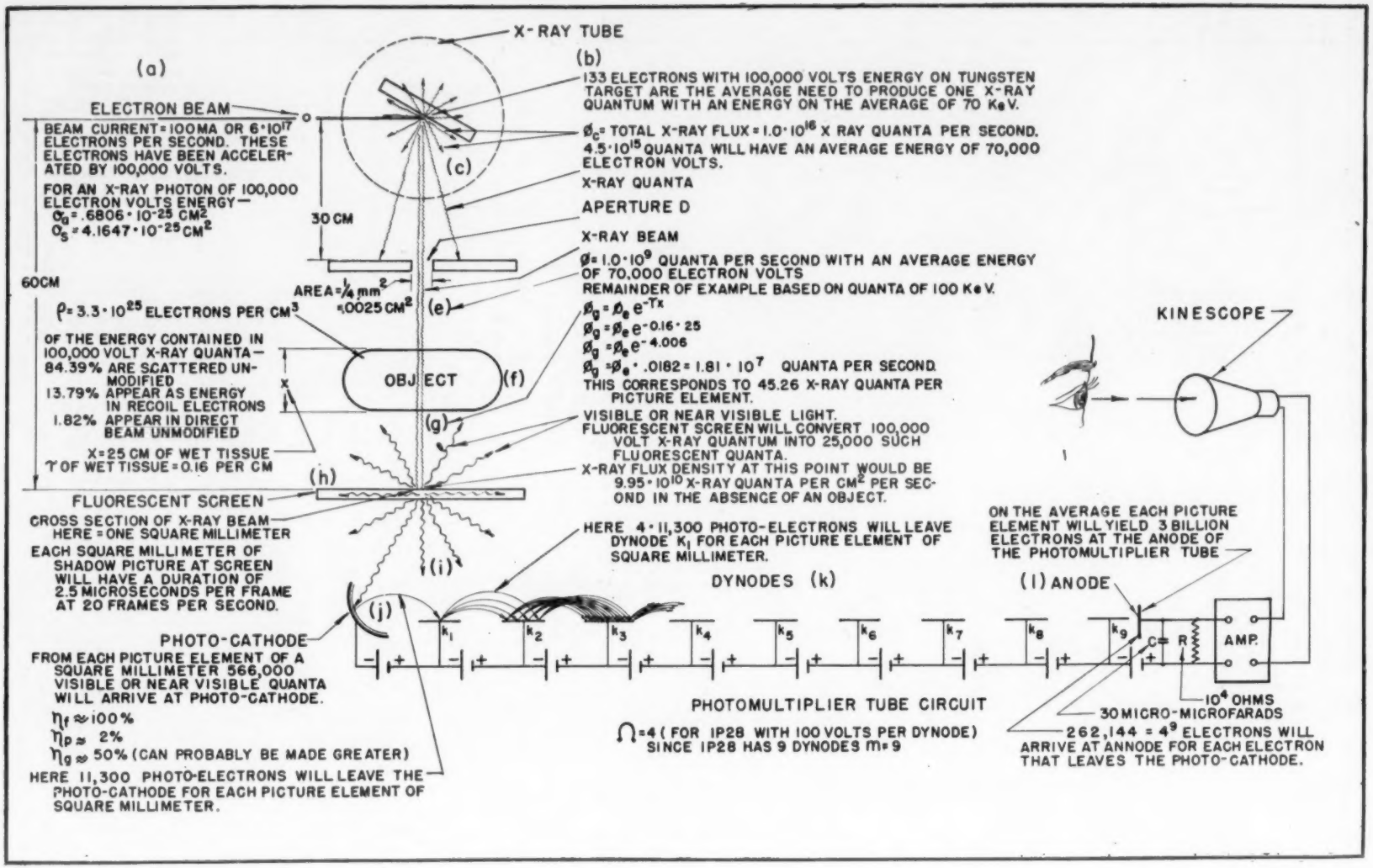
X-RAY TUBE
(a)
ELECTRON BEAM
BEAM CURRENT = 100 MA OR 6·10^17 ELECTRONS PER SECOND. THESE ELECTRONS HAVE BEEN ACCELERATED BY 100,000 VOLTS.
FOR AN X-RAY PHOTON OF 100,000 ELECTRON VOLTS ENERGY—
σ_a = .6806 · 10^-25 CM^2
σ_s = 4.1647 · 10^-25 CM^2
30 CM
60CM
AREA = 1/4 mm^2 = .0025 CM^2
ρ = 3.3 · 10^25 ELECTRONS PER CM^3
OF THE ENERGY CONTAINED IN 100,000 VOLT X-RAY QUANTA—
84.39% ARE SCATTERED UNMODIFIED
13.79% APPEAR AS ENERGY IN RECOIL ELECTRONS
1.82% APPEAR IN DIRECT BEAM UNMODIFIED
X = 2.5 CM OF WET TISSUE
τ OF WET TISSUE = 0.16 PER CM
OBJECT
FLUORESCENT SCREEN
CROSS SECTION OF X-RAY BEAM HERE = ONE SQUARE MILLIMETER
EACH SQUARE MILLIMETER OF SHADOW PICTURE AT SCREEN WILL HAVE A DURATION OF 2.5 MICROSECONDS PER FRAME AT 20 FRAMES PER SECOND.
PHOTO-CATHODE
FROM EACH PICTURE ELEMENT OF A SQUARE MILLIMETER 566,000 VISIBLE OR NEAR VISIBLE QUANTA WILL ARRIVE AT PHOTO-CATHODE.
η_f ≈ 100%
η_p ≈ 2%
η_g ≈ 50% (CAN PROBABLY BE MADE GREATER)
HERE 11,300 PHOTO-ELECTRONS WILL LEAVE THE PHOTO-CATHODE FOR EACH PICTURE ELEMENT OF SQUARE MILLIMETER.
(b)
133 ELECTRONS WITH 100,000 VOLTS ENERGY ON TUNGSTEN TARGET ARE THE AVERAGE NEED TO PRODUCE ONE X-RAY QUANTUM WITH AN ENERGY ON THE AVERAGE OF 70 KeV.
Ø_c = TOTAL X-RAY FLUX = 1.0 · 10^16 X RAY QUANTA PER SECOND. 4.5 · 10^15 QUANTA WILL HAVE AN AVERAGE ENERGY OF 70,000 ELECTRON VOLTS.
X-RAY QUANTA
APERTURE D
X-RAY BEAM
Ø = 1.0 · 10^9 QUANTA PER SECOND WITH AN AVERAGE ENERGY OF 70,000 ELECTRON VOLTS
REMAINDER OF EXAMPLE BASED ON QUANTA OF 100 KeV.
Ø_g = Ø_e e^-Tx
Ø_g = Ø_e e^-0.16 · 25
Ø_g = Ø_e e^-4.006
Ø_g = Ø_e · .0182 = 1.81 · 10^7 QUANTA PER SECOND.
THIS CORRESPONDS TO 45.26 X-RAY QUANTA PER PICTURE ELEMENT.
VISIBLE OR NEAR VISIBLE LIGHT. FLUORESCENT SCREEN WILL CONVERT 100,000 VOLT X-RAY QUANTUM INTO 25,000 SUCH FLUORESCENT QUANTA.
X-RAY FLUX DENSITY AT THIS POINT WOULD BE 9.95 · 10^10 X-RAY QUANTA PER CM^2 PER SECOND IN THE ABSENCE OF AN OBJECT.
HERE 4 · 11,300 PHOTO-ELECTRONS WILL LEAVE DYNODE K_1 FOR EACH PICTURE ELEMENT OF SQUARE MILLIMETER.
DYNODES (k)
(a) (b) (c) (d) (e) (f) (g) (h) (i) (j) (k) (l)
KINESCOPE
ON THE AVERAGE EACH PICTURE ELEMENT WILL YIELD 3 BILLION ELECTRONS AT THE ANODE OF THE PHOTOMULTIPLIER TUBE
(l) ANODE
K_1 K_2 K_3 K_4 K_5 K_6 K_7 K_8 K_9
C R AMP.
PHOTOMULTIPLIER TUBE CIRCUIT
10^4 OHMS
30 MICRO-MICROFARADS
Ω = 4 (FOR IP28 WITH 100 VOLTS PER DYNODE) SINCE IP28 HAS 9 DYNODES m = 9
262,144 = 4^9 ELECTRONS WILL ARRIVE AT ANNODE FOR EACH ELECTRON THAT LEAVES THE PHOTO-CATHODE.

FIG. 4

amount of roentgen rays were used in taking a picture of the gear and were of the order of one-tenth of a milliroentgen. It was obtained with 5 milliamperes of tube current and a tube voltage of 60 kilovolts plus the attenuation due to the transit of the roentgen rays through one mil of tantalum foil and a very poor angle of the roentgen rays in respect to the electron beam which had to be used in this particular model. These last two factors result in an attenuation of possibly six to one. It is planned to use a tube voltage of 125 kilovolts which should increase the yield of roentgen rays by a factor of four over that of the present tube. It is also planned to use a beam current of the order of 100 milliamperes. This would further increase the roentgen-ray yield by a factor of twenty. However, it is interesting to note that the amount of useful roentgen rays generated by such a tube and associated pinhole would still be less than the amount of useful roentgen rays generated per second by the present day tube which is used for roentgenoscopic examinations. It might also be added that if a tilted target type of scanning roentgen tube is used, a line focus principle can be employed since the point of generation of roentgen rays on the target will be viewed at a small angle with respect to the surface of the target.

I might say, as a physicist, I am indeed somewhat optimistic about the future development of a sufficiently potent scanning roentgen tube which will supply sufficient number of quanta in a narrow beam, and all indications seem to be that the next tube of the water-cooled type will yield forty-five quanta per square millimeter in two and one-half microseconds, the average time duration of a single picture element. If the one-fifth second integration time of the eye is taken into consideration, this will yield approximately two hundred roentgen-ray quanta incident on the fluorescent screen after passing through some 25 centimeters of wet tissue. This particular link, i.e. the number of quanta incident on the fluorescent screen after passing through the object, is the weakest point statistically in the whole system, but this number of quanta will make the statistical fluctuation between regions of 100 per cent contrast, namely, between that of no roentgen rays and that region where the roentgen rays have passed through 25 centimeters of wet tissue of the order of 7 per cent. Figure 3 states the general case for this type of system and Figure 4 the special case where the roentgen-ray beam is generated at a voltage of 100 kv. and a current of 100 ma. and the other factors have definite and known values.

In conclusion, I wish to thank the several persons who have worked with me these past three months so that we might today demonstrate to you the feasibility of this system. The success up to this point leads us to hope that we do have here something that can be developed into a practical means of amplifying the fluoroscopic image.

Dr. Fred J. Hodges, Ann Arbor, Michigan. Statistical fluctuation mentioned so prominently by Dr. Sturm explains why, following two critically searching discussions of these four papers, time should be granted for remarks by one who lays no claim to mathematical or physical training. Perhaps I more nearly represent the majority of this audience than any of the previous speakers because my great interest in this subject of fluoroscopic image intensification is bolstered by great faith in its ultimate accomplishments. In my vast ignorance of the technical and theoretical difficulties to be overcome, I refuse to look with anything but enthusiastic confidence toward their ultimate complete solution.

The enthusiasm shown by Sturm and Morgan belies the note of gloom which they have sounded. Their remarks remind me of my former physics professor. Before every demonstration of classical physical phenomena, he used to express fervent hope that the experiment would be successful, knowing full well that it always would. If they were in serious doubt regarding the practical success of screen image amplification, their present furious experimentation would be abandoned promptly.

One is reminded of the difficulties, apparently insurmountable, which confronted photofluorography as recently as the mid-thirties. Ten years of energetic design, development of screens, films and lens systems plus extensive road-testing have given us a thoroughly practicable new technique. I for one expect nothing less gratifying from the various researchers who are striving for intensification success. We are living in a period of remarkable technical improvements in the tools of our profession. Screen intensification appears to be one of the most promising and important of these.

DR. MORGAN (closing). The hour is late and I shall do my best to sum up as quickly as possible.

Dr. Moon's experiments with the scanning roentgen tube are indeed very gratifying. Mr. Sturm and the rest of our group, as short a time as two weeks ago, finished a modified Moon system in which we used the conventional roentgen tube and an old type television scanning disc. We did this because we didn't have available to us the excellent facilities of the Rauland Corporation which Dr. Moon has.

Like Dr. Moon we were able to see, on the screen, images of very much the same quality that you saw on the viewing screen here this afternoon. Certainly, it is a method that is worth following up. It is an entirely feasible system from the technical standpoint; from the standpoint of ease and simplicity of construction, it does not offer the benefits of the tube which Mr. Coltman has talked about this afternoon.

There is also one other serious limitation with Dr. Moon's system which should be called to your attention at this time. It will be noted in Dr. Moon's Figure 4 that the roentgen-ray flux density at his pick-up fluorescent screen is 10^{11} roentgen-ray photons per square centimeter per second in the absence of an object. Now with a fluorescent screen, 4 inches square in size, this means that each square millimeter of the screen will receive 10^5 roentgen-ray photons per second. Also if one fluoroscopes an abdomen in the anteroposterior projection, this will reduce the number of roentgen-ray quanta to only 10^3 photons per square millimeter per second. Now this is exactly the number of photons which stimulate the eye in conventional present-day fluoroscopy. Therefore, according to the fluctuation theory that has been presented by Mr. Sturm this afternoon, Dr. Moon's system at best may be expected to be only as good as a conventional fluoroscope. Furthermore, these results can be obtained in Dr. Moon's system only with a fluoroscopic roentgen-ray generator operating at a constant potential of 100 kv. and 100 ma. (i.e. 10 kilowatts).

It may be argued, of course, that fluctuation theory is incorrect in estimating the detail provided by the Moon system. However, experimental measurements, which we have made with the system in our laboratory, justify confidence that the calculations are reasonably precise.

The inherent limitations of the Moon system seem insurmountable at this time. The pinhole diaphragm which is a fundamental part of the system discards so much of the radiation available from the roentgen-ray tube that an insufficient amount of radiation is available for good image quality. Accordingly, it appears that unless some completely unforeseen development takes place in the Moon system, image tubes like that described by Mr. Coltman will have considerably greater application in clinical fluoroscopy.

In answer to Dr. Hodges, who stated that we had caused a feeling of gloom to fall over this audience by anticipating that gains of only 30 to 50 or 60 times would be beneficial in the screen intensification process, we did so for a purpose. There has been a general feeling of great optimism as a result of the experimental work which you have heard about this afternoon. Indeed many people who have talked with me in the past few months have wondered whether or not they should buy any new roentgen-ray equipment. They felt that the new apparatus will so quickly revolutionize the entire field that the equipment that they have now will soon be obsolescent. We felt that a note of caution was in order.

I believe that the data that have been presented are sound and, contrary to the impression that Dr. Hodges apparently has received, we believe that the outlook is extremely gratifying. I think that Mr. Sturm in his paper expressed that opinion.

A gain of 30 to 50 times in brightness is a notable achievement. It will, as you saw in some of the curves, make the great mass of fluoroscopy which we will be pursuing in the future, as good as x-ray film; furthermore, even under the worst conditions, the situation will be immeasurably better than the situation that we now have today where experimental measurements indicate, for example, that the smallest object that can be perceived at 10 per cent contrast is of the order of one centimeter in diameter.

Certainly, we may expect much better performance than that. I feel that now that we have sounded our word of caution, we should leave this meeting with a note of optimism. The future for screen intensification is certainly bright, and very real benefits from screen intensification systems may be expected in a few years.

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INFECTIOUS MONONUCLEOSIS*

WITH SPECIAL REFERENCE TO ROENTGENOLOGIC MANIFESTATIONS

By JAMES J. McCORT, M.D.

BOSTON, MASSACHUSETTS

THE first clinical description of infectious mononucleosis is credited to Filatow,[16] a Moscow pediatrician, in 1885. Apparently unaware of this report, in 1889 Pfeiffer,[26] a pediatrician in Vienna, again described it and named it glandular fever because of the clinical signs of fever and lymph node enlargement. Neither of these observers recognized any specific changes in the blood. In 1905 Burns[7] in Baltimore noted a peculiarity of the mononuclear cells in patients with glandular fever. His observations, as well as those made by European pediatricians, appeared to receive little attention during the ensuing years, and no series of cases were reported during the mobilization period of World War I. In 1920 Evans and Sprunt[14] published a report of 6 cases of what they called infectious mononucleosis and described the blood changes in detail. Several years later Tidy[35] in England called attention to the fact that the two entities, glandular fever and infectious mononucleosis, were identical. Knowledge of the blood changes was amplified by the reports of Longcope[23] and of Downey and McKinley.[11] Based on the earlier work of Forssman,[18] the discovery of Paul and Bunnell,[25] in 1932, that the serum of patients ill with the disease was capable of agglutinating the red blood cells of sheep in high dilution, the so-called heterophile antibody test, made available a ready and fairly accurate method of determining the presence of this disease.

PATHOLOGIC FINDINGS

Investigators have always been limited by a paucity of pathologic material due to the generally benign nature of infectious mononucleosis. In a study of 10 biopsied lymph nodes, Gall and Stout[19] found marked proliferative activity within the pulp and large numbers of specific infectious mononucleosis cells present throughout the nodes. The pathologic changes to be seen in the spleen, of which 9 were studied, were recorded by Smith and Custer.[32] The spleen, which was enlarged in 8 out of the 9 cases, contained many normal and atypical lymphocytes which infiltrated the trabeculae and in some cases completely replaced the follicles.

Du Bois[12] in 1930 recorded the first autopsy of a case of infectious mononucleosis, that of a patient who died of empyema. The lungs when sectioned showed a confluent bronchopneumonia and a purulent bronchiolitis. It was impossible, however, to determine which, if any, changes were due to the infectious mononucleosis *per se*.

Thomsen and Vimtrup[34] in 1939 found 6 fatal cases among a total of 500 cases of infectious mononucleosis; these were described in detail, 2 of them having had postmortem examination. In one of the latter in which death was due to degenerative changes in the respiratory center of the brain, the mediastinal and mesenteric lymph nodes were enlarged, while the lungs were found to be edematous and hyperemic. Enlargement of the mediastinal and mesenteric lymph nodes was present also in the second autopsied case which had been complicated by a pneumococcic septicemia; the lungs were negative on microscopic study. In both cases there was enlargement of the lymph nodes around the common bile duct. Unfortunately no roentgenographic examination was made in any of the 6 cases.

In 1942 Jersild[21] published a case report with postmortem examination of a patient

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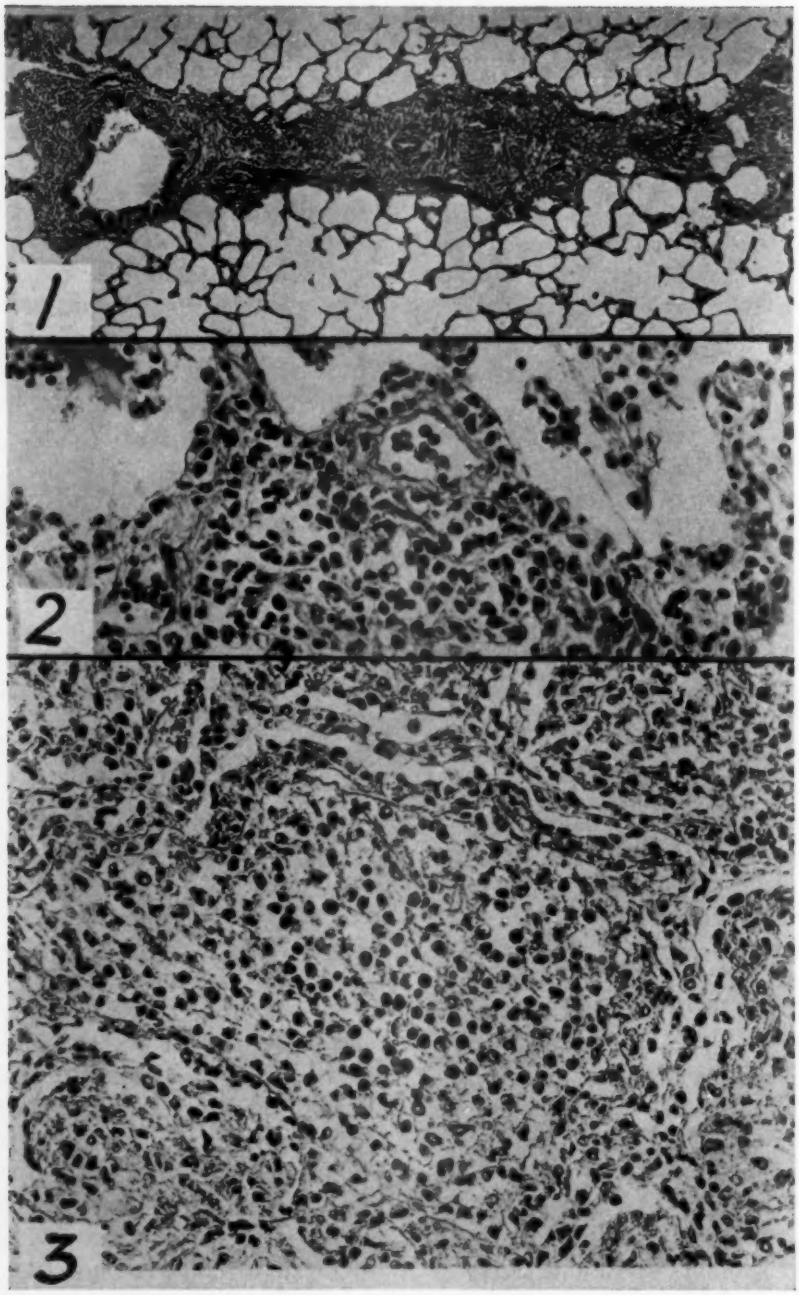

Fig. 1.* Lung. Peribronchial connective tissue and interlobar septum heavily "infiltrated" with lymphoid cells; the process involves intra-alveolar septa as well, rendering them thick and more prominent (×30).
Fig. 2.* Lung. Extensive interstitial pneumonitis with virtually no intra-alveolar exudate; the cellular reaction is entirely lymphocytic (×450).
Fig. 3.* Lung. Lobular pneumonia showing a rich fibrous network supporting a cellular exudate which is almost entirely of lymphoid type (×450).

* Figures 1, 2 and 3 reproduced though the courtesy of Custer and Smith,[8] from *Blood*.

in whom the diagnosis had been substantiated by blood smear and the heterophile antibody test. A diffuse myocarditis was found to be the cause of death; the lungs were described as normal.

Subsequently there appeared a report of a single autopsy by Ziegler[41] in 1944. His patient died of a ruptured spleen ten days after admission to the hospital. Microscopic examination of the lungs showed capillary distention with atypical mononuclear cells as well as scattered perivascular and interstitial foci of mononuclear cells. There was also a moderate infiltration of the bronchial walls by the mononuclear cells.

The next publication on postmortem changes in infectious mononucleosis was that of Allen and Kellner[2] which appeared in 1947. In this case, during an acute attack of infectious mononucleosis, roentgenologic examination of the chest showed changes which were described as "primary atypical pneumonia, etiology unknown." About three weeks later the patient, an Air Force pilot, was killed in an airplane crash, and at autopsy focal cellular infiltrations of infectious mononucleosis cells were found in the lungs as well as in the liver, kidneys, heart, adrenals, testes, and brain.

In the same year Ricker *et al.*,[31] on the basis of 2 cases studied clinically and at autopsy, called attention to the similarity of the neurologic manifestations of infectious mononucleosis to those of the Guillian-Barre syndrome, and postulated a probable causal relationship.

The most detailed study on the pathology of infectious mononucleosis to date is that of Custer and Smith[8] published in 1948. Their material consisted of 9 cases from the files of the Army Institute of Pathology which included the single case of Allen and Kellner as well as the 2 cases of Ricker *et al.* Analysis of the lung changes in the 9 cases showed distinct lesions in 3; minimal lesions in 5; and no pulmonary abnormalities in 1. In some instances atypical mononuclear cells were found in the intra-alveolar septa in the manner seen in interstitial pneumonitis (Fig. 1 and 2). Custer and Smith also found frank pneumonic consolidation in 2 cases; in one of which the cellular exudate was of the classical polymorphonuclear type while in the other the alveoli contained large numbers of atypical mononuclear cells (Fig. 3).

The most recently reported autopsy in infectious mononucleosis is that of Dolgopol and Husson[10] in 1949 whose patient died nine days after the onset of the illness with respiratory paralysis, presumably due to a central nervous system lesion. No enlarged nodes were found in the hilar regions; atypical lymphocytes were present in the interstitial tissues of the lungs, beneath the mucosa, and in the pleura, but there was no interstitial pneumonitis.

MATERIAL

During the past six years a diagnosis of infectious mononucelosis has been made on 75 patients at the Massachusetts General Hospital. Of this group 43 had roentgenologic examination of the chest during their illness. A review of these roentgenograms shows that in 6 patients there were changes which it is believed could be attributed to the disease. These 6 cases and an additional similar one encountered during military service form the basis of this study. Definite hematologic evidence is available in all 7 patients establishing the diagnosis of infectious mononucleosis. That the pulmonary changes to be discussed are due to the disease is not definitely proved since all 7 made a spontaneous recovery with complete regression of the pulmonary abnormalities. Of the 43 whose chests were examined during the acute stage of their illness, 38 showed enlargement of peripheral lymph nodes clinically and 4 of these enlargement of the paratracheal or peribronchial lymph nodes roentgenologically. This does not necessarily mean, however, that the mediastinal nodes were not enlarged in any of the other 34 cases but rather that they were not sufficiently enlarged to be perceptible on the chest roentgenogram. In 4 of the 43 examined

roentgenologically there was also present a pneumonitis which later cleared completely.

INCIDENCE OF AND CLINICAL FINDINGS IN INFECTIOUS MONONUCLEOSIS

The clinical picture of infectious mononucleosis has been described adequately in the excellent monograph of Bernstein[5] who summarized the world literature on the disease in 1940. The intervening period has been covered by Sturgis[33] in his textbook on hematology. Both authors found a definite age limit in patients suffering from this disease; it is one of children and young adults and is very unusual past the age of forty. The infection has a world wide distribution. It is said to be rare in the Negro race, but sporadic cases have been reported from time to time.[40] In general, the sex incidence is 3 to 2 in favor of males.

In a large series reported from an army post during World War II by Vander Meer *et al.*[37] the clinical manifestations appeared in the following order of frequency: malaise, cervical adenopathy, fever above 102°F., white blood cell count above 11,000, generalized aching, headache, and a spiking temperature curve. Milne[24] found sore throat a frequent complaint. On physical examination the lymphadenopathy is usually most striking although the nodes show only slight to moderate enlargement. They are discrete and often tender. Enlargement of the spleen, which is tender to palpation, occurs in about 50 per cent of the cases,[33] usually reaching to a few fingers-breadth below the costal margin, and in its distended condition may easily rupture. A note of warning against too vigorous palpation of this friable organ is sounded by Smith and Custer.[32] Palpation with its consequent danger may be avoided by roentgenography; a plain film of the upper abdomen, centered over the hypochondrium, gives adequate information. In the present group, 12 patients showed a splenic enlargement that was apparent on the chest roentgenogram.

In monocytic angina or the angiose type of infectious mononucleosis, examination may also disclose a tonsillar membrane or ulceration. Jaundice has been found in 9.1 per cent of 64 patients studied by Abrams.[1]

LABORATORY DATA

The diagnosis of infectious mononucleosis is usually suspected when the blood examination is complete and the blood smear studied. An initial polymorphonucleosis with as high as 75 per cent polymorphonuclear cells is said to be not infrequent.[36] During the course of the illness a monocytic and lymphocytic reaction follows, at which time there is an absolute rise of these cells with many atypical forms. The importance of the heterophile antibody test as corroborative diagnostic evidence has already been noted. This test is made more accurate by absorption tests with beef cells and guinea pig kidney.[17] The test does not become positive until several days after the onset of the disease or even later. Paul and Bunnell[25] are of the opinion that a positive reaction is proof of infectious mononucleosis and that a negative reaction does not exclude it. The heterophile antibody test is also positive in serum sickness. The frequent finding of a falsely positive Wassermann reaction during the active stages of infectious mononucleosis as in other infections has been noted.[33]

ROENTGENOLOGIC PULMONARY CHANGES

That enlargement of the mediastinal lymph nodes occurred in infectious mononucleosis was suspected by both Filatow and Pfeiffer, as shown in their early clinical descriptions of the disease, and later by others,[23] on the basis of the patient's nonproductive cough. It is reasonable to expect that a disease causing enlargement of the peripheral lymph nodes might bring about a similar change in the mediastinal and mesenteric lymph nodes. Not all cases of infectious mononucleosis present peripheral adenopathy, but when it is present, roentgenologic evidence of similar enlargement of the mediastinal nodes would not be unex-

pected. Unfortunately, roentgenographic examination of the chest is not a routine procedure in this disease, and the actual incidence of this finding is very low. Even when a chest examination is made, mitigating against the discovery of nodal enlargement is the fact that the mediastinum ment of the mediastinal lymph nodes (Fig. 4, 5 and 6).

Changes in the peripheral lung fields have been observed. Based on the studies of Custer and Smith these are believed to be due to an interstitial infiltration or an alveolar filling by the atypical lymphocytes,

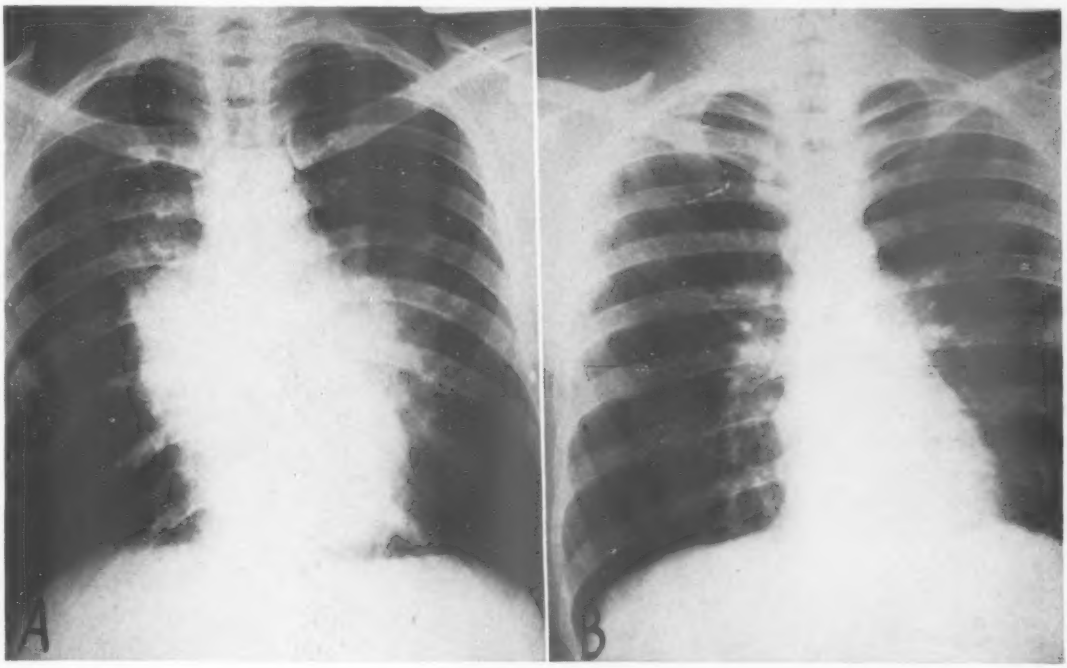

Fig. 4. W. J. S., a twenty-one year old white enlisted man, was admitted to an Army general hospital complaining of headache, cough and fever of 101°F., present for several days. Four days after admission the cervical, axillary and inguinal lymph nodes were found to be enlarged and the spleen was palpable. *Laboratory data:* Red blood cell count, 5,450,000; white blood cell count, 15,000, with 23 per cent polymorphonuclears, 73 per cent lymphocytes, 4 per cent monocytes, many prolymphocytes; hemoglobin, 17.4 gm. Heterophile antibody positive 1:512; Kahn test positive (? false positive); tuberculin test negative 1:10,000 forty-eight hours; coccidioidin test negative, forty-eight hours; sputum negative for acid fast bacilli.

(*A*) Roentgenogram shows a marked enlargement of peribronchial lymph nodes with a strand-like increase in density in both lungs. (*B*) Roentgenogram taken three months later shows complete clearing of the periphery and regression of the enlarged nodes. (Clinically recovery was complete.)

and superimposed structures are composed of dense tissues and the nodes, to be visible, must be greater than 2 to 3 cm. in diameter in order to project outside the margins of the vascular shadows. Press *et al.*[29] reported 3 instances of enlargement of the mediastinal nodes out of a group of 96; single instances in smaller groups have been described by others.[4,15,22,23] Of the 7 patients in the present series with roentgenologic changes, 5 showed detectable enlarge- although one case reported by them showed filling of the alveoli with polymorphonuclear cells. Several authors have reported isolated cases in which pulmonary changes were found. In a patient who recovered spontaneously, described by Erf,[13] there was bronchopneumonia in the lower lobes. In an early report of Tidy[35] a patient showed dullness at the bases during the course of the disease but no chest roentgenogram was made. In 1 case out of 65

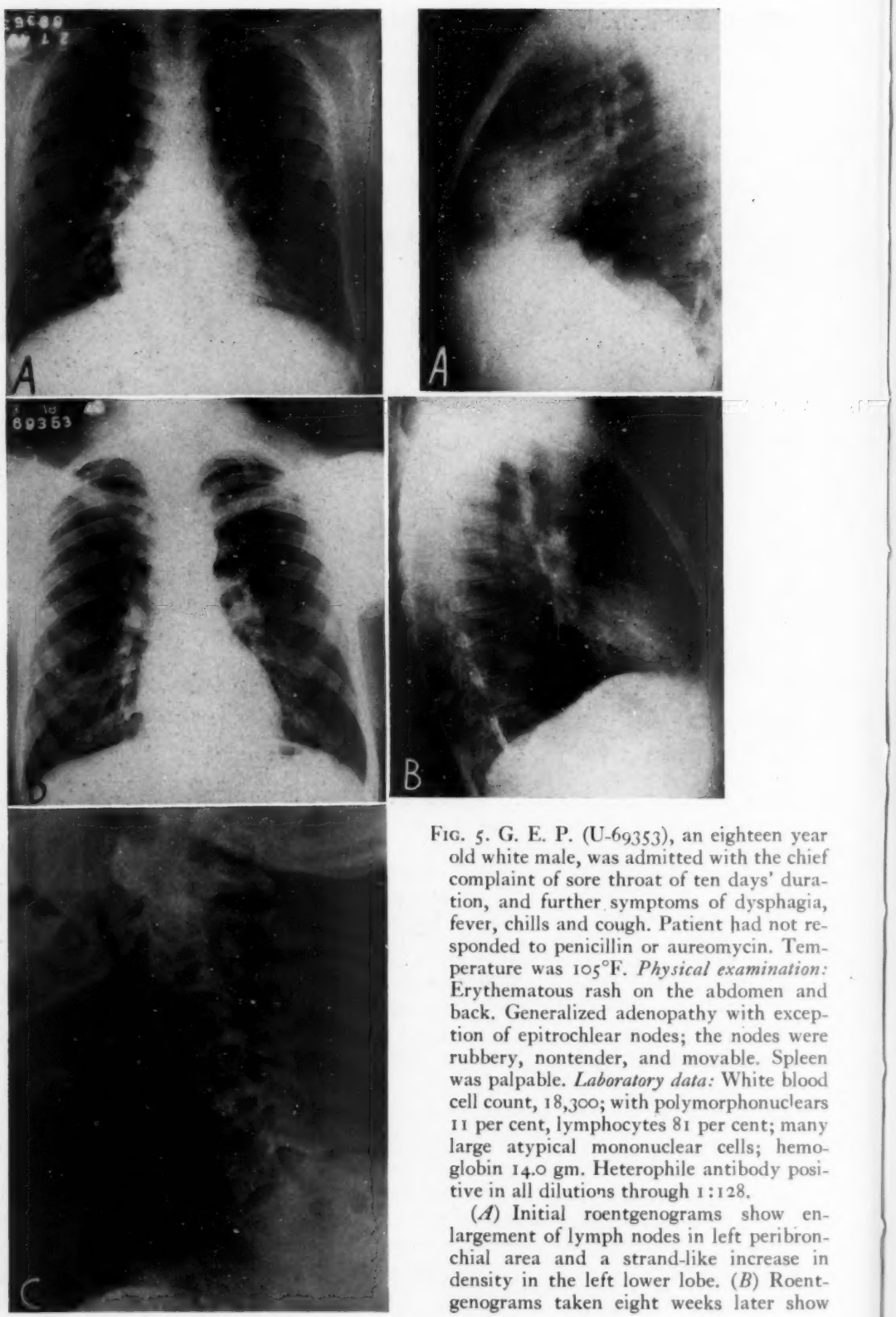

Fig. 5. G. E. P. (U-69353), an eighteen year old white male, was admitted with the chief complaint of sore throat of ten days' duration, and further symptoms of dysphagia, fever, chills and cough. Patient had not responded to penicillin or aureomycin. Temperature was 105°F. *Physical examination:* Erythematous rash on the abdomen and back. Generalized adenopathy with exception of epitrochlear nodes; the nodes were rubbery, nontender, and movable. Spleen was palpable. *Laboratory data:* White blood cell count, 18,300; with polymorphonuclears 11 per cent, lymphocytes 81 per cent; many large atypical mononuclear cells; hemoglobin 14.0 gm. Heterophile antibody positive in all dilutions through 1:128.

(*A*) Initial roentgenograms show enlargement of lymph nodes in left peribronchial area and a strand-like increase in density in the left lower lobe. (*B*) Roentgenograms taken eight weeks later show

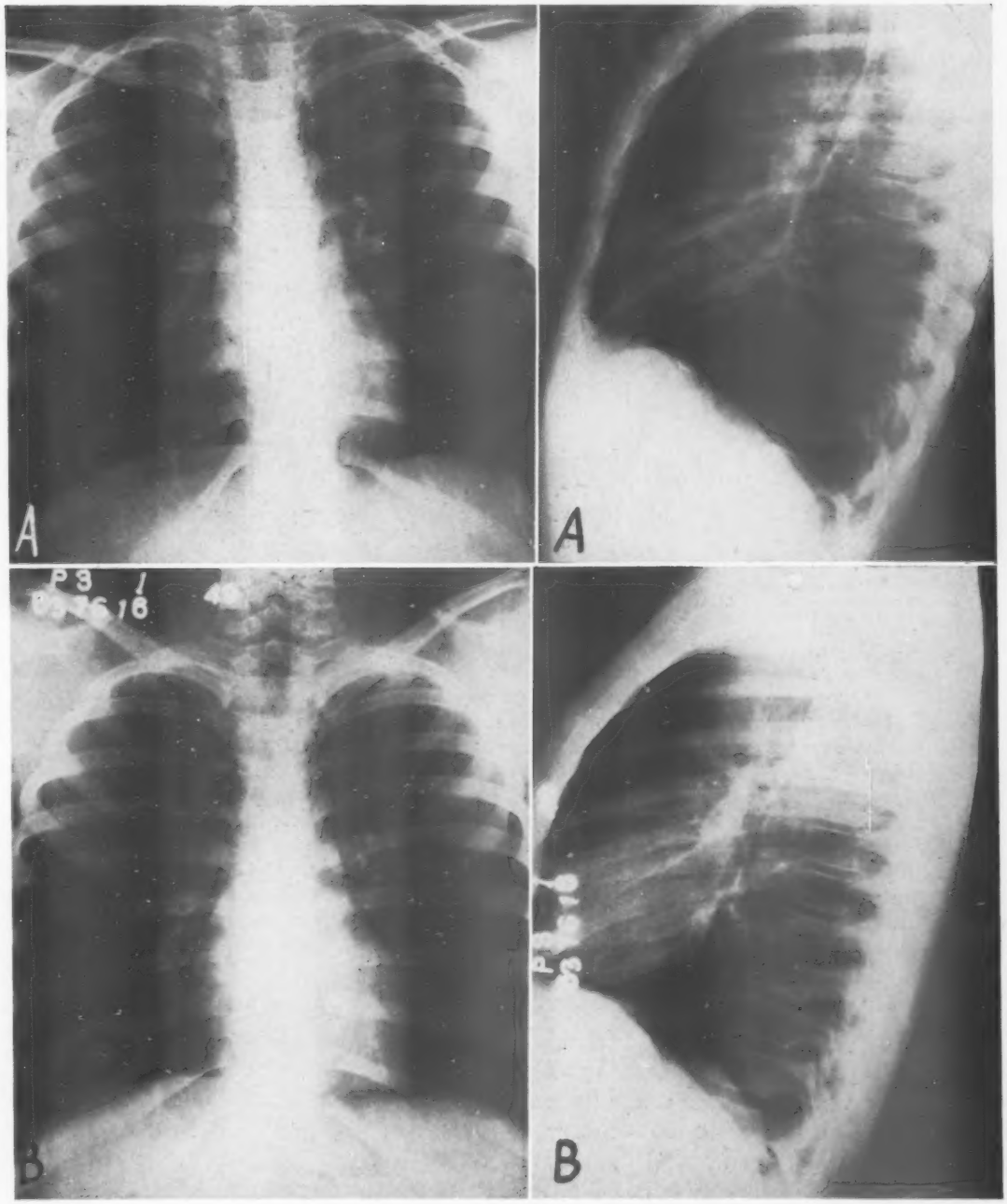

Fig. 6. M. D., Jr. (U-637616), a fifteen year old white male, was admitted complaining of malaise and difficulty in swallowing. *Physical examination:* Small tender lymph nodes in the cervical, axillary, inguinal and epitrochlear regions. The spleen was barely palpable. *Laboratory data:* White blood cell count, 17,000, with 66 per cent polymorphonuclears and 34 per cent lymphocytes. There were several atypical forms of both the lymphocytic and monocytic series. Heterophile antibody positive 2 plus in a dilution of 1:128.

(*A*) Roentgenograms on admission show enlarged lymph nodes in the left hilum; the lungs are otherwise clear. (*B*) Examination made five months later shows the lymph nodes to have regressed completely.

diminution in size of the lymph nodes and partial resolution of the process in the left lower lobe. (*C*) Lateral examination of the neck, made on admission, shows enlargement of the lingual tonsils and swelling of the epiglottis, which probably account for the complaint of sore throat and dysphagia.

seen at Johns Hopkins Hospital by Bernstein[5] clinical signs of bronchopneumonia were found. Boquien and Vercelletto[6] observed tiny nodular densities in the lung parenchyma during the course of the disease in 1 case; this seems to be unusual and no similar cases are known to have been reported. Two patients with clinical signs of pleurisy were seen in an "epidemic" of 84 cases reported by Raeburn,[30] and a sterile pleural effusion was found in a patient described by Power and Boucher.[28]

In the previously mentioned autopsied case of Allen and Kellner,[2] as well as in 14 out of 556 cases of Wechsler *et al.*,[39] roentgen changes in the lung were described as "similar to primary atypical pneumonia, etiology unknown." The roentgenologic similarity is understandable on the basis of the pathologic studies of Custer and Smith, which indicate that in some cases the microscopic changes of interstitial infiltration can be caused by atypical lymphocytes and monocytes in infectious mononucleosis (see Fig. 1 and 2).

In 5 of the 7 cases in this study changes were found in the pulmonary parenchyma (Fig. 4, 5 and 7). In 3 of these the parenchymal changes were associated with mediastinal lymph node enlargement. In none of the 5 patients did the clinical course bear a great resemblance to atypical or bacterial pneumonia, nor was the pneumonitis suspected clinically. It was an incidental finding on the chest roentgenogram, and its true character is unproved; it is possible, however, that it represents pulmonary changes associated with the infectious mononucleosis process. In this small group the speed of resolution of the pulmonary changes varied,—in one patient the lungs were entirely clear one week after the initial examination; in another it was very slow, the lungs not clearing completely until three months after the initial roentgenogram.

An interesting report by Poppel and Starr[27] appeared in 1942. In one of their cases small pressure defects were seen on the wall of the second portion of the

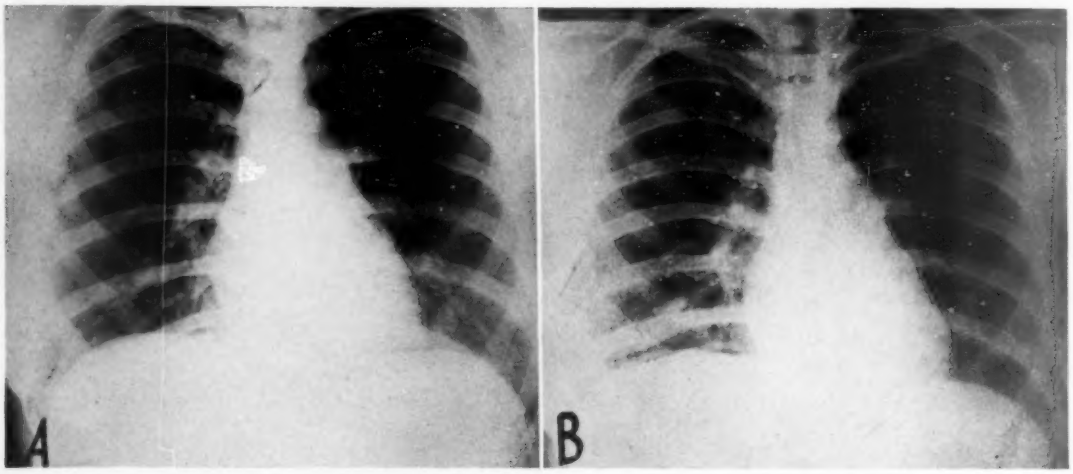

Fig. 7. E. T. (U-453411), an eighteen year old white student nurse, developed malaise one week before admission and five days before complained of generalized aches and pains, and anorexia followed by headache, nausea and cough. *Physical examination:* Patient was feverish but in no acute distress. Enlarged lymph nodes were palpable on both sides of the neck.

(*A*) Examination of chest on admission shows a pneumonitis in the left base. *Laboratory data:* White blood cell count, 10,400, with 52 per cent polymorphonuclears, 38 per cent large lymphocytes, and 6 per cent small lymphocytes. Heterophile antibody 1 plus in dilution of 1:32. (*B*) Chest examination one week later shows complete resolution of the pneumonitis in the left base. *Laboratory data:* White blood cell count, 17,000, with 20 per cent polymorphonuclears, 2 per cent large lymphocytes, 36 per cent small lymphocytes and 1 per cent monocytes, atypical lymphocytes 34 per cent. Heterophile antibody positive 1:64.

duodenum; this they attributed to lymph node enlargement although they were unable to prove this hypothesis. Similar defects were seen in one of the cases in this series. These findings gain importance in the light of the 2 autopsies reported by Thomsen and Vimtrup[34] in both of which enlargement of the lymph nodes surrounding the common duct was found.

DIFFERENTIAL DIAGNOSIS

Very little difficulty is encountered in establishing the diagnosis of infectious mononucleosis. An adenopathy occurring in a young person with splenomegaly, fever, malaise, and sore throat usually raises the suspicion, which is strengthened by an abnormal blood smear, and confirmed by a rising blood titer of sheep red blood cell agglutinins. The roentgen evidence of mediastinal lymphadenopathy must be differentiated from that associated with other diseases, particularly lymphoma, leukemia, sarcoid, erythema nodosum, coccidioidomycosis, and primary tuberculosis. This differentiation can be made only by correlation with the clinical history and findings. As previously mentioned, the pulmonary parenchymal changes resemble atypical pneumonia or other virus infection. Perhaps in the future a therapeutic trial of aureomycin may be of value as a differential procedure.

COMPLICATIONS

Infectious mononucleosis, a benign, self-limited disease, has very few complications. Of these the most formidable is that of splenic rupture, and this is responsible for the majority of deaths so far reported in the literature. Other complications that have been recorded are hemolytic anemia,[3] thrombocytopenic purpura,[9,40] central nervous system involvement,[10,31,36,38] myocarditis,[21] and hepatitis with jaundice.[1] Relapses occur in a certain number of cases, and a chronic state of the disease has been described.[20]

SUMMARY

Of seventy-five cases of infectious mononucleosis treated at the Massachusetts General Hospital, forty-three have had roentgen examination of the chest during the course of the disease.

In seven cases of clinically proved infectious mononucleosus, roentgen examination showed the following:

1. Enlargement of the mediastinal lymph nodes in two cases.
2. Both enlargement of the mediastinal lymph nodes and pulmonary parenchymal changes in three cases.
3. Parenchymal changes only in two cases.

Positive changes on a chest roentgenogram in infectious mononucleosis are infrequent. On the basis of published pathologic studies, it is suggested that pulmonary changes when present are due to engorgement of the hilar lymph nodes and infiltration of the interstitial tissue of the lung by atypical lymphocytes and monocytes.

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THE ANGIOCARDIOGRAPHIC DIAGNOSIS OF SYPHILITIC AORTITIS*†

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SYPHILITIC aortitis may be considered the fundamental lesion of cardiovascular syphilis in that saccular aneurysm, aortic insufficiency and coronary artery disease develop secondarily to it. As might be expected, uncomplicated syphilitic aortitis is also the commonest form of cardiovascular syphilis. Moore estimates that 70 to 90 per cent of all patients with late syphilis detectable at postmortem examination show evidence of cardiovascular damage, usually syphilitic aortitis.[1]

Saccular aneurysm, insufficiency of the aortic valve and coronary ostial atresia can be altered relatively little by antisyphilitic therapy, but the application of specific therapy to uncomplicated aortitis can be expected to result in arrest of the inflammatory process and prevention of more serious lesions.[2] The ideal management of cardiovascular syphilis is generally agreed to be its detection and treatment in the stage of uncomplicated aortitis, and considerable effort has been expended to improve the means for early diagnosis. The yearly physical examination of the patient with late latent syphilis is mainly for the purpose of detecting the earliest signs of involvement of the cardiovascular system.

CONVENTIONAL METHODS OF DIAGNOSIS

Uncomplicated syphilitic aortitis is rarely productive of symptoms. Aortalgia, a burning substernal pain, has been described, but its inconstancy makes it a most unreliable criterion. Physical signs are of little more diagnostic value since they can be produced by hypertension and arteriosclerosis, both of which commonly occur in the same age range as syphilitic aortitis. Emphasis has been placed on the quality of the aortic second sound, a tambour-like accentuation being pointed to as evidence of aortitis. Similar sounds are so often produced by arteriosclerosis and hypertension as to render this sign of doubtful differential value. Percussion of the chest is also unreliable in that widening of the area of supracardiac dullness occurs rarely in uncomplicated aortitis and usually indicates the presence of aneurysm.

Roentgenography and roentgenoscopy of the chest have been of considerable value in detecting cases of uncomplicated aortitis and differentiating them from other types of cardiovascular disease.[3] Calcification of the ascending aorta has been found to be a relatively reliable sign of syphilitic aortitis; however, its presence is not pathognomonic and its absence is of no diagnostic significance. Obvious widening of the aorta is significant, but does not occur frequently enough to be used as a diagnostic criterion and is usually a late manifestation by the time it becomes evident on conventional roentgen examination. The portion of the aorta most commonly affected by syphilis is the ascending portion which is hidden by the heart in the conventional chest roentgenogram. Various measurements of aortic width have been shown to be indirect, inaccurate and inapplicable to the root of the aorta where the earliest manifestations of syphilitic aortitis occur.[4,5]

A positive serological test for syphilis, a history of antisyphilitic therapy or collateral evidence of syphilis on physical examination, plus widening of the aorta, calcification of the ascending aorta, evidence of

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aneurysm or the presence of the diastolic murmur of aortic insufficiency have been considered sufficient to make a diagnosis of cardiovascular syphilis. It has been difficult to detect early cases of syphilitic aortitis and occasionally to differentiate between syphilitic and hypertensive or arteriosclerotic disease.

ANGIOCARDIOGRAPHY

Angiocardiography affords exact visualization of the gross structure of the thoracic aorta during life. Utilizing the method of Robb and Steinberg,[6] the angiocardiographic characteristics of syphilitic aortitis were studied. It was found that in the presence of clinical or roentgen evidence of aortitis, and occasionally in their absence, certain angiocardiographic signs were regularly present. These signs, critically evaluated in 60 cases, included aortic dilatation, irregularity of aortic lumen, calcification of the ascending aorta, aortic tortuosity, abnormal aortic wall thickness, and aneurysm.

Dilatation. Dilatation is the most significant angiocardiographic evidence of syphilitic aortitis, varying from slight enlargement of the ascending aorta to widespread dilatations or saccular aneurysm. A previous report has outlined in detail a method for measurement of the angiocardiographically visualized aorta.[5] Briefly, these measurements are made in the left anterior oblique projection at four points: (*A*) the mid-ascending aorta, (*B*) the transverse aorta, (*C*) the mid-descending aorta, and (*D*) the diaphragmatic aorta (Fig. 2*b*). Measurements in this series of 60 cases of syphilitic aortitis were compared to corresponding measurements of a hundred normal aortas. A summary of the results is presented in Table I, and a graphic comparison of the caliber of the normal and syphilitic mid-ascending aorta may be seen in Figure 1. Measurements of the ascending aorta in the presence of syphilitic aortitis in 51 of our cases varied from 38 to 70 mm., and averaged 45.4 mm. in caliber, as compared to a normal range of from 16 to 38 mm. with an average of 28.6 mm., all ages being considered. In syphilitic aortitis, the transverse, descending and diaphragmatic measurements were similarly higher than the corresponding normal figures, but the incidence and degree of dilatation diminished as the point of measurement left the proximal aorta, a fact in agreement with the pathological evidence that the ascending aorta is the earliest affected in syphilitic aortitis.

TABLE I

TABLE SHOWING COMPARATIVE MEASUREMENTS MADE ON 100 NORMAL AORTAS (INCLUDING ARTERIOSCLEROSIS) AND 60 CASES OF SYPHILITIC AORTITIS.

Aorta	Normal			Syphilitic		
	No. Cases	Range mm.	Average mm.	No. Cases	Range mm.	Average mm.
Ascending	100	16–38	28.6	51	38–70	45.4
Transverse	44	13–34	24.8	39	25–49	32.3
Descending	58	12–32	22.9	30	24–50	31.3
Diaphragm	54	9–28	19.7	18	20–43	28.2

In the presence of syphilis and in the absence of other detectable causes for aortic widening, dilatation of the aorta may be considered diagnostic of syphilitic aortitis. However, it is not in itself pathognomonic of syphilitic aortic disease. We have repeatedly observed abnormal dilatations of the ascending aorta (over 38 mm.) in the presence of hypertension, rheumatic aortic

valvular insufficiency and coarctation of the aorta. We have never observed aortic dilatation above the upper limit of normal which could be attributed to arteriosclerosis alone. Hypertension and syphilis often co-exist, occasionally making the interpretation of aortic dilatation difficult. No cases were included in this series when other potential causes of aortic dilatation cast doubt upon the diagnosis of syphilitic aortitis. Conversely, the absence of aortic dilatation does not exclude the presence of aortitis since histopathologic changes in the aortic media occur in the absence of gross aortic dilatation early in the course of the disease. In assembling this series of cases, several cases with clinical evidence suggesting syphilitic aortitis were excluded in the absence of angiocardiographic evidence of aortic dilatation. Two of these excluded cases in the middle age group had calcification of the ascending aorta and probably had syphilitic aortitis.

Irregularity of Lumen. Non-syphilitic aortic dilatation tends to be uniform and smooth in outline. The dilated, unfolded lumen of the hypertensive aorta shows diffuse rather than local dilatation, and although as in syphilis the maximum dilatation is that of the mid-ascending aorta, the transition to a normal width occurs gradually throughout the entire aortic arch. In syphilitic aortitis, the caliber of the aorta may be seen to vary in a more irregular fashion, a finding of definite differential value. The presence of an irregular aortic lumen was noted in 51 (95 per cent) of our cases.

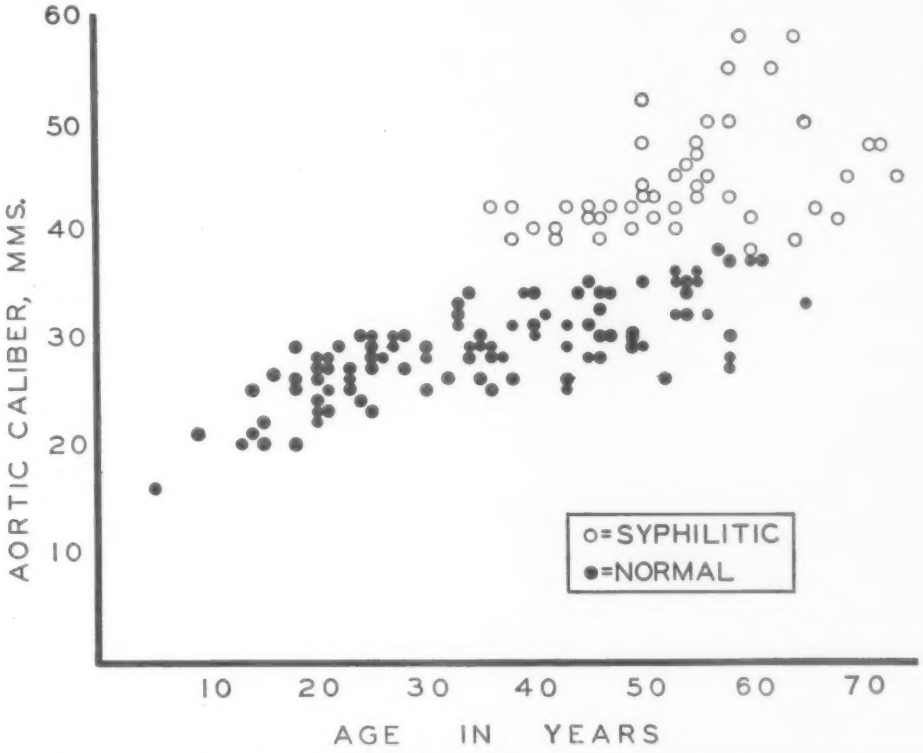
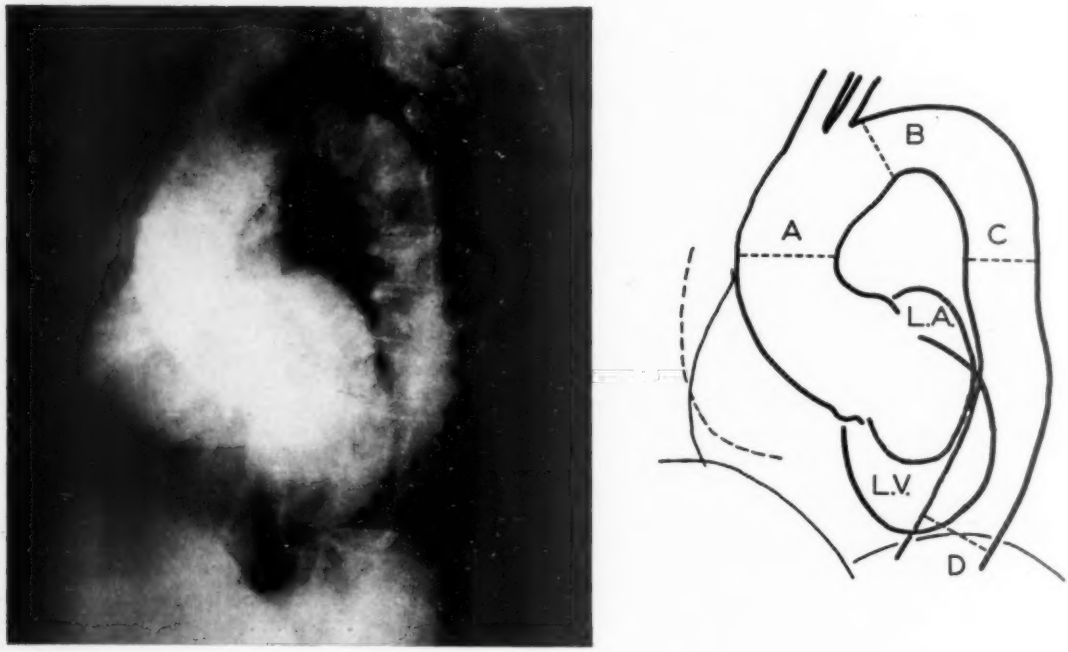


Fig. 1. Graphic representation of caliber of mid-ascending aorta in 100 normal aortas (solid black dots) and 46 cases of syphilitic aortitis (circles). Arteriosclerosis considered as "normal."

Calcification of the Ascending Aorta. In this series, roentgenographic evidence of calcification of the ascending aorta was present in 16 cases (26.6 per cent), a figure comparable to that reported by Jackman and Lubert[7] in an analysis of 66 proved cases of syphilitic aortitis. We have observed calcification of the ascending but

not the descending aorta in instances where there was no clinical or angiocardiographic evidence of syphilis. The absence of calcification of the ascending aorta is thus of no significance in ruling out syphilitic aortitis, while its presence may be considered presumptive but not pathognomonic evidence of the disease.

wall as demonstrated angiocardiographically were occasionally striking, these findings, on the whole, are regarded as inconclusive. Atherosclerotic plaques cause abnormal aortic wall thickness. Assuming the normal aortic wall thickness to be approximately 2 mm., we have observed abnormally thick and thin walls at and between



a *b*

Fig. 2*a*. Angiocardiographic study of aorta (left anterior oblique projection) of a fifty-three year old female upon whom the clinical but not the roentgen diagnosis of syphilitic aortitis had been made. Noté dilatation, irregularity, and tortuosity of ascending aorta and aortic arch. No calcification was present.

Fig. 2*b*. Tracing of Figure 2*a*. Routine sites of measurement: (*A*) mid-ascending (43 mm.); (*B*) transverse (27 mm.); (*C*) mid-descending (30 mm.); (*D*) diaphragmatic (32 mm.).

Aortic Tortuosity. In 51 (95 per cent) of our cases, there was demonstrable tortuosity of the aorta. Since the age groups of syphilis and arteriosclerosis are widely overlapping and since arteriosclerosis generally produces tortuosity of the aorta, this finding is of relatively little value. It becomes of considerable diagnostic significance, however, in the younger age group, as does the presence of calcification in the aortic arch, regardless of its location.

Variations in Aortic Wall Thickness. Although variations in thickness of the aortic areas of dilatation in aortitis. Aneurysm walls were seen to vary between 1 and 15 mm. in thickness, the latter figure probably reflecting the combined presence of laminated thrombus and scar tissue.

Aneurysm. Aortic aneurysms, while occasionally non-syphilitic in nature, are generally regarded as diagnostic of aortic syphilis. Angiocardiography makes the recognition of aortic aneurysm a relatively simple procedure. Of our 60 cases, 25 patients had aortic aneurysms, of which 7 were multiple aneurysms, 18 were saccular

aneurysms, 7 were fusiform aneurysmal dilatations. Arbitrarily, we consider a diffuse fusiform dilatation of the aorta aneurysmal if its maximum caliber exceeds 60 mm.

DISCUSSION

In revealing clearly the gross anatomy of the entire thoracic aorta, angiocardiography greatly facilitiates the diagnosis of syphilitic aortitis. The earliest and most constant angiocardiographic sign of syphilitic aortitis is dilatation of the ascending aorta which although highly suggestive is not pathognomonic since it may be caused by other pathologic processes, notably hypertension and rheumatic aortic insufficiency. In the presence of syphilis and in the absence of hypertension or rheumatic aortic valvular disease, the diagnosis of syphilitic aortitis may be made angiocardiographically even in the absence of clinical or conventional roentgenographic signs. Early dilatation of the ascending aorta cannot be detected with regularity by conventional roentgenographic means. The angiocardiographic diagnosis of syphilitic aortitis in the absence of aortic dilatation should not be made. Other commonly encountered angiocardiographic evidences of syphilitic aortitis include irregularity of the aortic lumen, calcification in the ascending aorta, aortic tortuosity, variations in aortic wall thickness and aneurysm. A typical case of syphilitic aortitis is illustrated in Figure 2, *a* and *b*.

Since angiocardiography is occasionally inadequate diagnostically, the diagnosis of syphilitic aortitis should always be based upon a careful evaluation of history, physical findings and roentgen examination. In many instances, contrast visualization is of confirmatory value only. In at least one-third of the cases in this series there existed reasonable clinical doubt and inadequate roentgen evidence of syphilitic aortitis. Aortas appearing unremarkable on conventional chest roentgenograms have repeatedly been shown to be dilated by angiocardiography. Contrast aortic visualization represents the most practical and accurate diagnostic technique for the early recognition of syphilitic aortitis.

CONCLUSIONS

1. Angiocardiography greatly facilitates the early diagnosis of syphilitic aortitis; with this procedure, a diagnosis can often be made in the absence of conclusive clinical or roentgen evidence.

2. The angiocardiographic signs of syphilitic aortitis include aortic dilatation (above 38 mm., mid-ascending aorta), irregularity of lumen, calcification of ascending aorta, aortic tortuosity, variations in aortic wall thickness, and the presence of aneurysm.

3. Dilatation, the cardinal angiocardiographic sign of syphilitic aortitis may also be caused by hypertension, aortic valvular insufficiency of non-syphilitic etiology, or coarctation of the aorta; it is not pathognomonic of syphilis of the aorta. Aortic dilatation may be absent in early cases of syphilitic aortitis. Uncomplicated arteriosclerosis apparently does not cause aortic dilatation beyond the upper limit of 38 mm.

4. By means of angiocardiography, the diagnosis of syphilitic aortitis may be made sooner and put on a sounder anatomic basis, thus facilitating the earlier, more adequate treatment occasioned by the cardiovascular complications of syphilis.

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DISCUSSION

Dr. Marcy L. Sussman, New York, N. Y. Dr. Dotter and his associates have made a really notable contribution to the study of syphilitic aortitis. They pointed out that in the absence of aneurysm and aortic insufficiency, the diagnosis is almost entirely a roentgenologic one. The history or serologic evidence of lues is the occasion for the roentgenogram being taken or confirms an incidental roentgen finding.

Although some workers have felt that aortic measurements from conventional films are of value in determining dilatation, others, including myself, feel that they are unreliable in early disease when they must be used as the deciding factor. In making this statement, I do not mean to detract from their use as a guide on the road to diagnosis. If we assume that the early treatment of syphilitic aortitis is desirable and important, Dr. Dotter's report concerning the precise appearance of the aorta is obviously worthy of note.

Dr. Dotter has told us that dilatation is the most significant angiocardiographic evidence of luetic aortitis. He reports that in 51 of his cases, the ascending aorta measured between 37 and 70 mm. while no normal measured more than 38 mm. We are in the midst of accumulating similar data but since I did not know precisely at what points Dr. Dotter made his measurements nor what corrections he made for the position of tube and patient to film, I am not in the position to critically evaluate this figure. We can anticipate, however, that in common with other statistical experience, when a larger series is accumulated, apparently normal individuals will be found in whom this measurement is exceeded.

This will not invalidate the test since, in any case, it is folly to rely on a single finding. Furthermore, as Dr. Dotter points out, there are other causes of dilatation. More important, however, are those aortas whose measurements are less than 38 mm. which nevertheless have dilated from a small normal. On a purely statistical basis, we would expect a number of these. By coincidence, if you saw two of the slides I showed yesterday, they illustrate these points. In the typical luetic aneurysm that I showed, the ascending aorta measured 36 mm. The dissecting aneurysm, which as you know is practically never on a luetic basis, had an ascending aorta measuring 44 mm. but that patient had hypertension.

It seems to me that irregularity in the caliber of the aorta when affected by lues is of paramount importance. We pointed this out some time ago when we found the sign of great value in cases of aortic aneurysm where the aneurysm did not fill with diodrast. Here the diagnosis of aneurysm could still be made when the remainder of the aorta showed an irregularity in caliber.

However, in this connection, a word of warning is in order. We are encountering an increasing number of distorted aortas with uneven constrictions and dilatations which we presume to be congenital in origin. We call the condition atypical coarctation because there are no resulting disturbances in circulatory dynamics. These peculiar aortas are found not infrequently in association with congenital aortic stenosis, subclavian and carotid stenosis, tetralogy of Fallot and other congenital anomalies, but sometimes no other abnormality has been detected.

Therefore, one cannot assume that luetic aortitis is present simply because there is an uneven lumen. Once again, I congratulate Dr. Dotter and his co-workers on doing well a job that needed badly to be done.

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INTRAVENOUS UROGRAPHY IN THE STUDY OF VESICAL NECK OBSTRUCTIONS*

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THERE are certain essential facts which should be known about patients with vesical neck obstruction in order to facilitate proper treatment and to increase the safety of operation. In addition to a careful general examination and routine laboratory studies, which all patients should have, a detailed appraisal of the status of the urinary tract is imperative. This should include: a determination of renal function and the condition of the upper urinary tract; detailed knowledge of the nature of the obstruction and the size and type of prostatic enlargement, as well as the presence of possible associated urinary tract disease, which may complicate the management. With intravenous urography we have a means of obtaining this information by a single examination, and this should be routine in all cases. Its value will be readily appreciated if we recall that it replaces less accurate renal function tests, and in most cases eliminates the need for cystoscopy and retrograde pyelograms; possibly urethrograms and cystograms as well. This is important inasmuch as some of these procedures are attended with definite risk, not to mention the discomfort they may provoke.

We have adopted the following routine in doing intravenous urograms which we particularly recommend for these cases. A preliminary blood urea estimation is performed, as we have found that if the urea is 70 mg. per cent or above, the urogram is usually unsatisfactory. The patient is preferably prepared the evening before by administering castor oil, and fluids are limited the following morning preceding the examination. A plain film of the abdomen is made followed by the intravenous administration of diodrast.

Before injecting the diodrast it is a wise precaution to inquire into any history of allergy or drug sensitivity in the patient, particularly to iodine. In doubtful cases one should perform a preliminary test by intradermal injection, or by placing 1 or 2 cc. of diodrast beneath the tongue. Although fatalities due to severe allergic reaction have been reported following the intravenous administration of diodrast, we have observed no serious reaction in the course of many thousand injections.

Following the injection of the dye 14 by 17 inch films of the entire urinary tract are made at intervals of five minutes, fifteen minutes, thirty minutes, and sixty minutes. At the time of the thirty minute film an additional 10 by 12 inch film is made to include the lower ureters, bladder, prostate, and urethra. This affords a more detailed cystogram. Following the last film, if desired, a residual urine film may be obtained after the patient has voided, thereby making it possible to ascertain the amount of urine remaining in the bladder.

The films are viewed as a series and their interpretation will be discussed under four headings: (1) renal function, (2) determination of the type of prostate, (3) the upper urinary tract changes and their significance, and (4) associated urinary tract lesions.

RENAL FUNCTION

Recent studies in renal physiology have shown that the ability of the kidneys to excrete diodrast (the diodrast clearance) is one of the most reliable tests of total renal function because this substance is cleared both by filtration and tubular excretion. The intravenous urogram may be considered as a visual means of estimating diodrast excretion. The films afford a frac-

* From the Cleveland Clinic and the Frank E. Bunts Educational Institute. Presented at the Forty-Ninth Annual Meeting, American Roentgen Ray Society, Chicago, Ill., Sept. 14–17, 1948.

tional function test which constitutes an additional refinement.

Findley[1] and his co-workers studied the iodine excretion in the urine following the intravenous injection of diodrast and compared it with the urea clearance test. They found that the normal kidneys excrete 40 to 50 per cent (average 45 per cent) of the diodrast in the first thirty minutes following injection and reductions in the rate of iodine excretion are roughly proportional to the variations in the urea clearance. This affords us a sound basis for the spacing of the films and serves as a guide in estimating the renal function when studying the films.

Other studies have been carried out to compare the urogram with other renal function tests. Kornblum and Fetter[2] found 70 per cent agreement with the fractional phenolsulfonphthalein test, and Braasch and Emmett[3] found 78 per cent agreement with the indigo carmine test.

While the intravenous pyelogram may lack the accuracy of the more refined renal function tests, one obtains a gross estimate of renal function which is entirely adequate to evaluate the surgical risk and direct the preoperative management. Only in those cases in which inadequate visualization renders functional estimation impossible have we found it necessary to resort to other renal function tests.

What criteria should be employed in evaluating the functional status of the kidneys from the interpretation of the films? With normal renal function there will be a prompt thrust excretion resulting in a good nephrogram and distinct visualization of the renal pelves in the five minute film. Maximum visualization of the upper urinary passages will occur in the fifteen and thirty minute films with only traces of dye remaining in the one hour plate. Additional evidence of adequate renal function is to be found in the increasing amount and intensity of dye in the bladder which reaches its peak on the one hour film.

At the other extreme let us consider the patient who, after the injection of diodrast, shows no trace of the dye throughout the urogram series with, perhaps, only traces in the bladder after one hour. This is indicative of definitely impaired renal function, and such a patient requires careful preoperative preparation.

Between these extremes numerous gradations may be observed. There are cases with delayed excretion but with satisfactory visualization of the upper urinary tract in the fifteen or thirty minute films which present no evidence of urostasis or dilatation and minimal retention of dye in the renal pelves in the one hour film. From the surgical point of view this indicates adequate renal function, and if the blood chemistry is within normal limits one may proceed with operation without the delay of prolonged drainage.

If, however, there is delayed function with maximum visualization on the thirty minute or one hour film which reveals dilated, tortuous ureters and hydronephrosis, impaired renal function is indicated and one must institute prolonged drainage until renal function is restored to a more normal level (Fig. 10).

Several observations are of interest. In general, the degree of renal damage is dependent upon the duration and type of obstruction. In this connection it is paradoxical that the longer the duration of symptoms and the larger the residual urine, the smaller the prostate. Thus the large, palpable, chronically overdistended bladder often occurs as the result of a fibrous contracture of the vesical neck or a small subcervical middle lobe. On the other hand, large intravesical prostates and subvesical hypertrophies, presumably of long standing, may present little if any renal damage from back pressure. Residual urine per se is no index of renal damage, for it is not uncommon to see patients with residual urines of 500 cc. or more with an essentially normal upper urinary tract.

Not all cases with diminished renal function can be attributed to vesical neck obstruction. With patients in the prostatic age group there will be a certain number with arteriosclerotic kidney disease and other renal lesions resulting in impaired function. Additional renal function studies

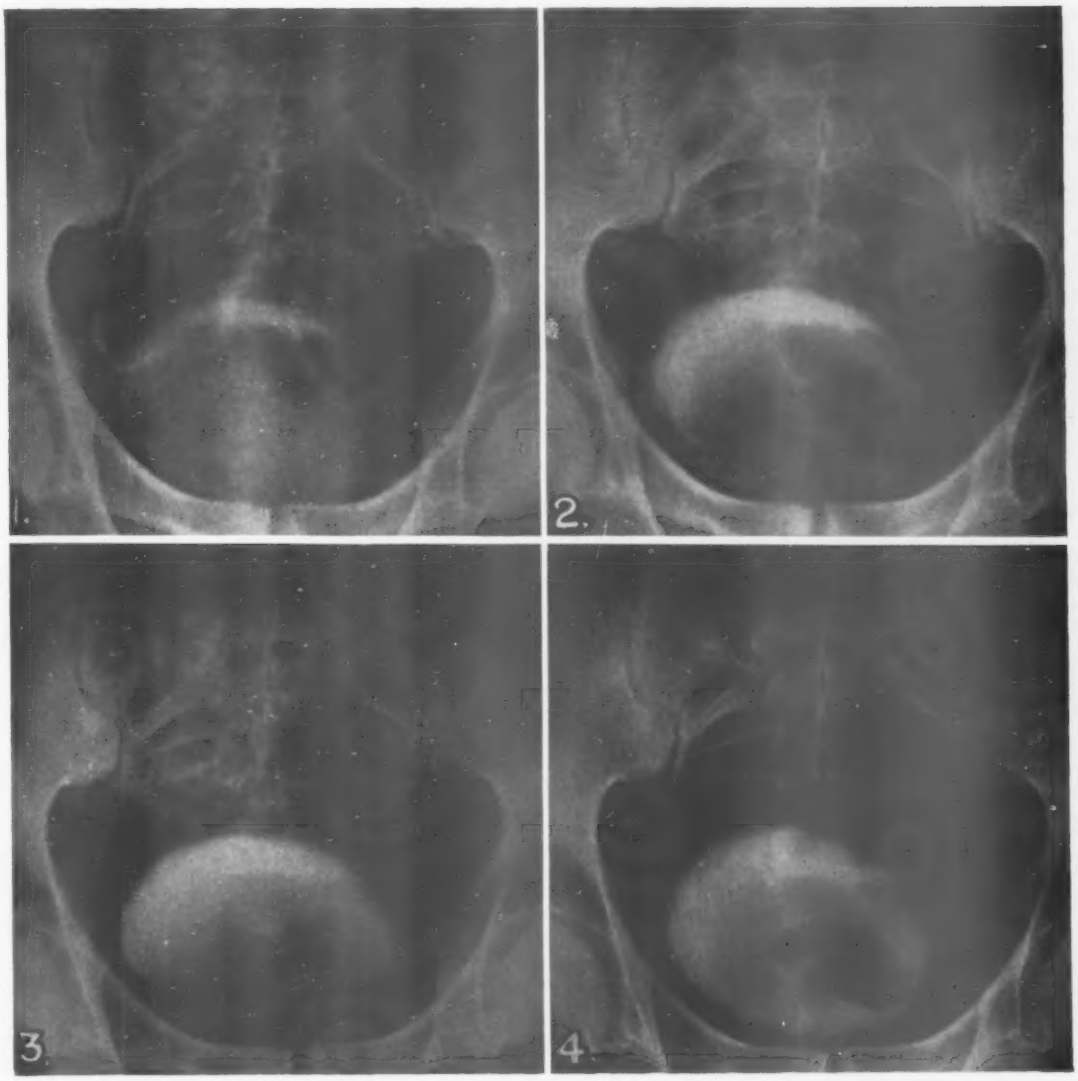

FIG. 1. Serial cystograms obtained by intravenous urography. No. 1 urogram five minutes; No. 2, fifteen minutes; No. 3, thirty minutes; No. 4, sixty minutes following diodrast injection. As bladder fills the prostatic shadow becomes obscured.

may be indicated in such cases, but the urogram will have served an important role in identifying those due to obstruction.

TYPE OF PROSTATE

Vesical neck obstructions fall into four types: (1) contractures and fibrotic median bars; (2) intravesical enlargement; (3) subvesical or intraurethral enlargement, and (4) carcinomas. Types 2 and 3 may exist in combination.

The serial cystogram affords valuable assistance in the differentiation of the various types. The urogram is started with the bladder empty and as the bladder fills visualization of deformities of the bladder base will be well demonstrated (Fig. 1). The roentgenological findings are dependent upon the type of obstruction and its resulting effect upon the bladder and upper urinary tract.

(1) Fibrotic contracture or median bar is a slow, insidious obstructing lesion of the vesical neck. The increased emptying effort of the bladder results in hypertrophy of the muscle, particularly the trigone. In this

Fig. 2. No. 2 urogram illustrating crescentic shadow produced by hypertrophy of trigone.

process there is often formed a deep depression behind the interureteric ridge, the so-called bas-fond, and in long-standing cases there is extensive trabeculation of the bladder with multiple sacculations or diverticula. Because there is no enlargement of the prostate the cystogram shadow will present no deformity of the bladder base. The hypertrophy of the trigone may be recognized by a transverse crescentic shadow whose concavity is toward the dome of the bladder (Fig. 2). In some cases the deep bas-fond will produce a circular or oval shadow of increased density lying within the cystogram(Fig. 3). These changes are best demonstrated in the fifteen or thirty minute film and may be obliterated with the bladder filled in the one hour film. The one hour film, however, best demonstrates the irregularities produced by sacculations and diverticula. In my experience, upper urinary tract changes come late with vesical neck contractures and are usually associated with decompensation of the bladder and large residual urine.

(2) Intravesical Enlargement. In 1922 Tandler and Zuckerkandl[4] studied the anatomy of benign prostatic hypertrophy and distinguished two types of enlargement both of which arise from the region of the prostatic urethra lying between the verumontanum and the vesical sphincter. In one, the prostate grows up through the bladder orifice and acquires an intravesical position. It is covered only by mucous membrane and the sphincter through which it grows often compresses it into a pedunculated lobe (Fig. 4). As it extends anteriorly it may assume a horseshoe shape or a variety of other shapes may result from different arrangements of adenomas (Fig. 5). From the standpoint of urographic interpretation, however, all have in common the intravesical position and some degree of narrowing of the base. Since they are able to expand within the cavity of the bladder, upper urinary tract changes are

Fig. 3. No. 3 urogram from patient with vesical neck contracture. Oval shadow within bladder shadow is produced by deep depression above and behind the vesical trigone.

not seen in the uncomplicated intravesical enlargement. I have on one occasion removed an intravesical hypertrophy weighing 330 gm. in which the intravenous urogram revealed a normal upper urinary tract.

The intravesical enlargements are thus recognized by the filling defect projecting into the bladder cavity from the floor of the bladder. This is characterized by the fact that the base is narrower than the main body of the projecting mass. This narrowing is due to the constricting effect of the internal vesical sphincter.

(3) Subvesical Enlargement. In the second type of prostatic enlargement described by Tandler and Zuckerkandl the whole tumor mass is subvesical. It is covered above by the bladder muscle and mucosa and the entire floor of the bladder is elevated (Fig. 6). The vesical orifice and sphincter, however, remain intact. Since expansion below is rendered impossible by unyielding ligaments and fascia, upward expansion progresses with resulting lengthening of the prostatic urethra and upward displacement of the trigone of the bladder.

Fig. 4. Drawing showing intravesical projection of enlarged prostate. (After Tandler and Zuckerkandl.[4] Taken from *Acta radiol.* supp. 28, 1935.)

In the urograms this produces two characteristic changes. The elevated bladder

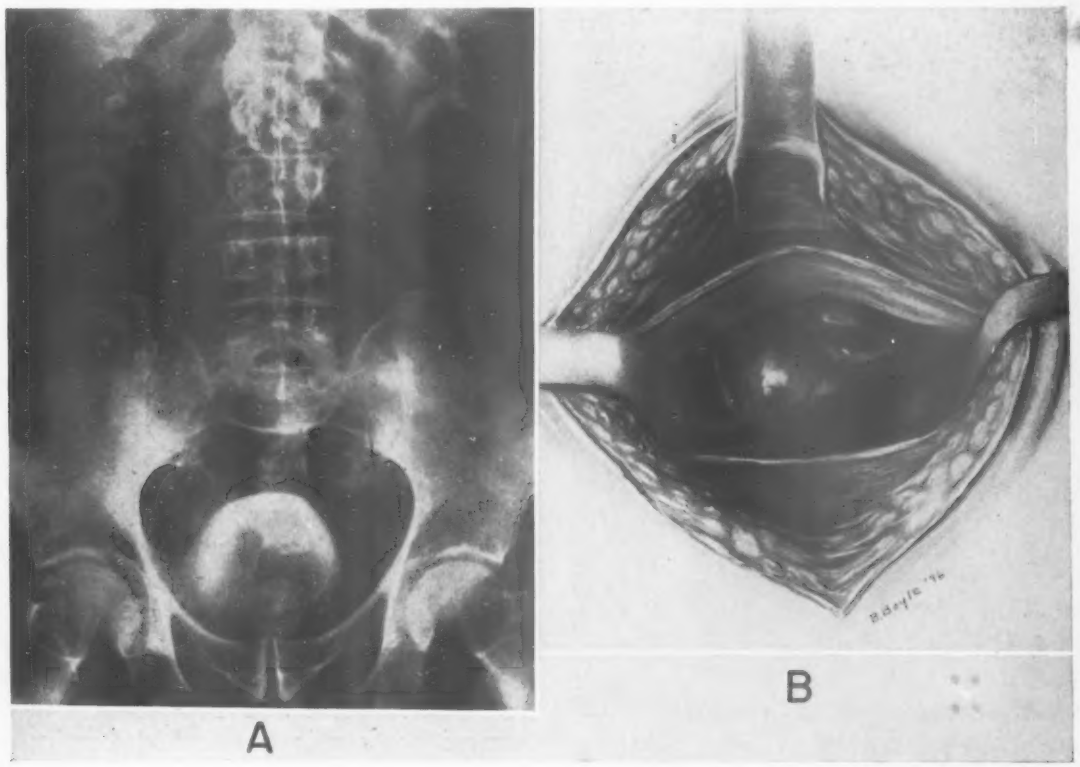

Fig. 5. (*A*) No. 3 urogram showing irregular intravesical prostatic shadow. Observe that the base is more narrow than the intravesical portion. (*B*) Drawing made at operation in same case showing multilobular intravesical prostatic enlargement.

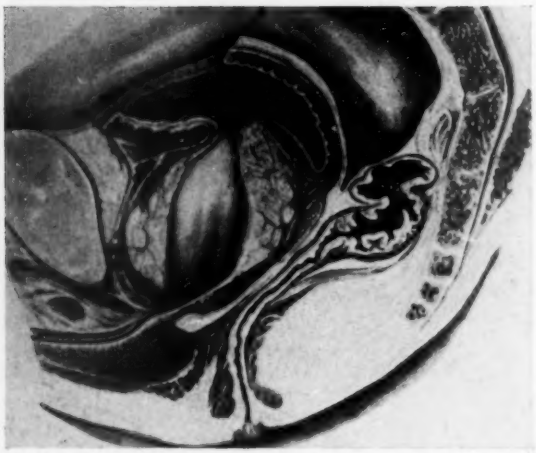

Fig. 6. Drawing showing subvesical enlargement of prostate. The entire bladder base is displaced upward. (After Tandler and Zuckerkandl.[4] Taken from *Acta radiol.*, supp. 28, 1935.)

Fig. 7. No. 3 urogram showing broad, dome-shaped prostatic shadow typical of subvesical hypertrophy. Note also "fish-hook" deformity of lower left ureter.

base manifests itself as a broad dome-like deformity whose base is its broadest point (Fig. 7). This is often best visualized in the early films of the cystogram series and may be obscured as the bladder fills around it (see Fig. 1). The second change is the result

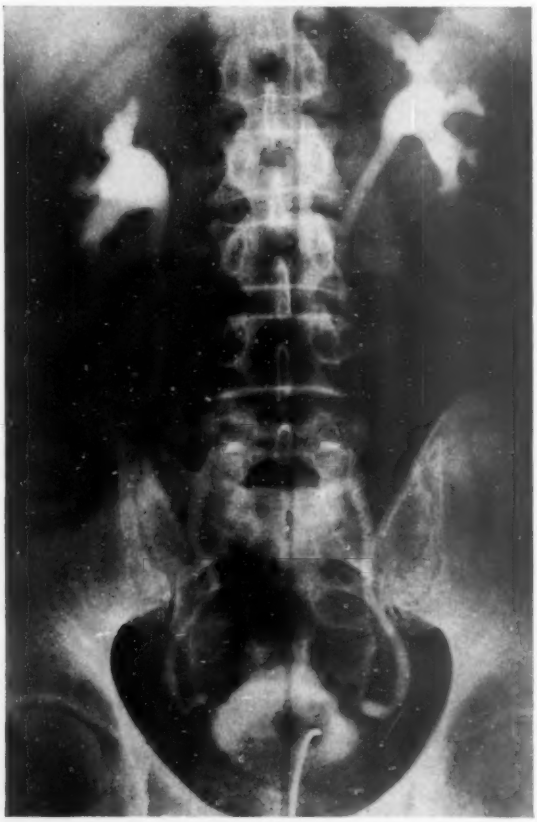

Fig. 8. No. 3 urogram illustrating bilateral "fish-hook" deformity of lower ureters. In this case early dilatation of the ureters and renal pelves is also demonstrated.

of the upward displacement of the trigone and ureteral orifices which produces angulation of the lower ureters of varying degree. In extreme cases a reverse curve is produced which, because of its shape, has been termed "fish hooking" (Fig. 7 and 8). The significance of this will receive later comment. Squire and Kretschmer[5] called attention to this ureteral deformity in prostatic hypertrophy but did not comment upon the mechanism of its production.

Combinations of intravesical and subvesical enlargement often occur in the same patient which may give rise to some difficulty of interpretation. Application of the broad principles enunciated above will usually lead to a correct assessment if one recalls that when the two coexist the intravesical mass "sits on top" of the subvesical enlargement. However, in some cases, the roentgenological findings may require the aid of the examining finger of the urologist or perhaps cystoscopic investigation to supply the needed information.

(4) Carcinoma of the Prostate. In the absence of bone metastases there are no consistent urographic findings which supply incontrovertible evidence of the presence of prostatic carcinoma. Suspicion should be aroused, however, if the prostatic shadow is irregular in outline or if distinctly irregular nodules are visible at the vesical neck. Also the presence of unilateral ureteral obstruction is suggestive evidence (Fig. 9). This is produced by unilateral extension of the neoplasm with invasion of the tissues surrounding the lower ureter. One cannot, however, expect much roentgenological assistance in the early diagnosis of carcinoma of the prostate. The trained examining finger is far more reliable.

STATUS OF THE UPPER URINARY TRACT

From the point of view of the prostatic surgeon, this is one of the most important considerations, and it is best determined by intravenous urography.

It should be pointed out that blood chemistry studies alone cannot be depended upon to give a clear picture of the status of the upper urinary tract. We have encountered patients with a normal blood urea in whom the intravenous urogram revealed pronounced bilateral hydroureter and hydronephrosis. Thus, the urogram is the more reliable guide to the status of the upper urinary tract.

Vesical neck obstruction is a slow insidious process which begins as pathological physiology and progresses to anatomical change with later renal functional impairment. At first the obstruction is overcome by increased contraction of bladder muscle which leads to hypertrophy of muscle and later trabeculation. Eventually, when the bladder is no longer able to empty itself, residual urine accumulates and the bladder may be said to be decompensated. There comes a time when the intravesical pressure approaches and then exceeds the ureteral secretory pressure and when this stage is reached, ureteral dilatation begins and gradually extends up to the renal pelves and collecting tubules, at which time impaired renal function results. This train of events is particularly applicable to chronic incomplete obstructions such as fibrous contractures and small obstructive intravesical lobes. In the subvesical hypertrophies an added factor of ureteral compression and

Fig. 9. No. 2 urogram showing dilatation of lower right ureter in a patient with carcinoma of prostate. Left ureter is normal.

angulation hastens the process of upper urinary tract dilatation.

Ureteral angulation should therefore be regarded with some concern. We have observed several patients who upon first examination presented the picture of moderate prostatic intrusion with early angulation of the lower ureters but with normal

Fig. 10. No. 4 urogram showing advanced bilateral hydronephrosis with dilated and tortuous ureters. Careful preoperative drainage is essential in such cases.

ureters and renal pelves. Re-examination of the same patients who returned after several years without treatment showed extensive bilateral hydroureter and hydronephrosis with impaired renal function. Ureteral angulation, therefore, should be regarded as an indication for the operative correction of prostatic obstruction if further upper urinary tract damage is to be avoided.

Bilateral upper urinary tract dilatation with delayed and impaired renal function is of significance to the urologist as an imperative indication for careful preoperative drainage (Fig. 10). Following an appropriate period of drainage, a recheck urogram will show striking recovery of renal function and a reduction in upper urinary tract dilatation. Only then does the patient become a safe surgical risk.

Kretschmer and Squire[6] recently studied the incidence of hydronephrosis in prostatic obstruction; 44.4 per cent of patients examined between 1933 and 1937 showed some degree of upper urinary tract dilatation while only 33.4 per cent of those observed between 1945 and 1947 were so affected. This difference was attributed to patients having sought earlier relief from obstructive symptoms. The average duration of symptoms was 60.5 months in the first group but only 43 months in the latter. Our experience coincides with this and today extensive bilateral hydronephrosis and ureterectasis is infrequently encountered.

Associated Urinary Tract Disease

Incidental but valuable information gathered from the routine use of urography for prostatic obstructions concerns the disclosure of additional and often unsuspected disease in the urinary tract. The plain film may reveal calculi in the kidneys, bladder or prostate, and in one instance a calculus impacted in the prostatic urethra had produced obstructive symptoms indistinguishable from bladder neck obstruction. By means of the urogram we have been able to distinguish renal tumors and solitary cysts. In one patient a congenital solitary kidney was recognized, while another showed renal tuberculosis. The cystogram shadow will indicate the presence of bladder diverticula and sacculations, and we have recognized several bladder tumors by the deformity produced in the cystogram series.

While this additional information may not always influence the treatment of the case, the incidental findings may prove to be the patient's most serious problem. In all instances this knowledge contributes to

intelligent diagnosis and consequent treatment.

SUMMARY AND CONCLUSIONS

Attention is drawn to the value of intravenous urography in the study of patients with obstruction of the urinary bladder neck and its routine use is recommended.

By means of this single examination we may determine (1) renal function, (2) the type of obstruction and the degree of enlargement of the prostate, (3) the status of the upper urinary tract and (4) associated urinary tract disease.

From this information the urologist is guided in (1) the preoperative treatment of the patient, (2) selection of operation and (3) evaluation of the risk. This knowledge, properly applied, increases the safety of prostatic surgery.*

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* For discussion, see page 673.

DEFORMITIES OF THE BLADDER SECONDARY TO URETERAL STONE*

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EDEMA and swelling about the ureteral orifice has long been recognized by the cystoscopist as a secondary sign of a calculus located in the intramural portion of the ureter. Actual prolapse of the mucous membrane of the ureter may occur as well. These changes are completely described and illustrated in many of the standard works on urology. Lowsley and Kirwin[3] state that cystoscopic examination will usually give definite evidence of the presence of a stone at or near the ureteral orifice. There will be swelling and edema which is sometimes accompanied by hemorrhage from the bladder mucosa. MacAlpine[5] describes the edema as first involving the immediate vicinity of the ureteral orifice, and later spreading to the interureteric bar, and the region of the trigone. In severe cases the whole trigone may be involved with the production of bullous edema.

Although these findings are well known to the cystoscopist, there has been very little attempt to correlate this observation with changes in the roentgenogram. Polgar[7] in 1936 reported one case in which he observed a filling defect in the region of the left ureteral opening secondary to a stone in the intramural portion of the ureter. He felt that in the plain roentgenograms of his case the urine alone was sufficient contrast material to outline the area of edema and swelling. However, he was able to demonstrate it better by means of cystography using a fairly dilute contrast material. Herskovits,[2] in 1938, following Polgar's description reported 2 cases with defects in the bladder due to the swelling about a calculus in the intramural portion of the ureter. These were observed in the course of excretory urography. Edling[1] in his article on the "Interureteric Fold in the Roentgenogram" mentioned the changes occurring in the interureteric region of the bladder as a result of calculi in the distal end of the ureter. No reference to this finding in the English journals was found after a fairly careful search. Kerr and Gillies' text on "The Urinary Tract," page 176, refers to Edling's paper but states they have so far found it of no value.

In the past seven years (1941–1948) at the Charles T. Miller Hospital, 106 patients in whom a diagnosis of ureteral stone has been established have had intravenous urograms. In 26 of the 106 cases, the stone was located in the intramural portion of the ureter. In 19 of these, deformities were present in the bladder shadow in the region of the ureteral orifice, undoubtedly due to edema and swelling caused by the stone. These were classified into three groups depending upon the degree of deformity of the bladder shadow. Four were classified as showing a marked deformity, 6 as moderate, and 9 as a slight but definitely recognizable deformity. Cystoscopic examination was done on 12 of the patients and in all of these the finding of edema and swelling of the region of the ureteral opening was confirmed.

The filling defects in the bladder are of two general types depending on the extent of the edema. First, those in which the swelling is confined to the region of the ureteral orifice and the interureteric ridge. In these cases, the filling defect is within the bladder shadow, in the anteroposterior view. There may be asymmetry of the interureteric ridge (Fig. 1 and 2). When the bladder is completely filled, as at the completion of urography or by retrograde cystography, the deformity is often less prominent as it becomes obscured by the dense may make it impossible to obtain clear outlines of the antrum. This statement applies

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contrast medium. Polgar[7] states that in this type, the deformity in the bladder contour can be better seen in the oblique projection. In this way a tangential view is obtained demonstrating the projection of the swollen ureteral orifice region into the bladder. In the second type of deformity, the edema and swelling is more pronounced and extends downward toward the trigone, or upward toward the dome of the bladder, or both, and results in a marked irregularity of the superior, inferior, or lateral portions of the bladder outline (Fig. 3, 4 and 5).

CLINICAL SIGNIFICANCE

In our experience, if the ureteral calculus is located in the intramural portion of the ureter, a filling defect in the bladder can be recognized in a large number of the cases (19 of 26 cases) and is therefore a common secondary sign of ureteral calculus. Calculi in this location are often tiny and difficult to diagnose, and any aid in their recognition is of value. This difficulty in diagnosis was emphasized by Peterson and Holmes[6] in 1937. They found, in reviewing 100 cases of ureteral calculi, that of 17 calculi not diagnosed on initial interpretations, 16 were located in or close to the intramural portion of the ureter.

Fig. 1. Asymmetry of the interureteric ridge with marked enlargement of the right side of the ridge extending to the lateral bladder wall (black arrows). Reddening and well marked edema about the right ureteral orifice was reported at cystoscopic examination. White arrow points to small stone.

Fig. 2. During the course of the intravenous urogram, an area of decreased density within the bladder shadow is visible (arrows). This surrounds the shadow of the stone in the ureter. At cystoscopy a pyramidal area of edema was present about the ureteral orifice.

If the calculus is non-opaque, its location and presence are difficult to establish roentgenographically. In these cases of non-opaque stones which are located in the intramural portion of the ureter, the filling defects of the bladder may be helpful as an aid in diagnosis and localization.

The filling defect within the bladder and the changes in its contour are often quite marked, and may simulate the defects secondary to bladder carcinoma. One of our cases (Fig. 4) demonstrates this very well. The entire left side of the bladder shadow is obliterated and the margin of the bladder shadow on the left is irregular. On the initial examination this defect was considered to be due to a carcinoma of the

FIG. 3. There is a filling defect on the right side of the bladder with asymmetrical enlargement of the interureteric ridge on the right. A calculus is faintly visible in the center of the defect (white arrow). At cystoscopy a marked edema of the right side of the bladder was visualized.

FIG. 4. There is marked irregularity and deformity of the left side of the bladder shadow. A calculus is visible in the lower left ureter overlying the coccyx (arrow). The stone was located at cystoscopy but could not be removed. It passed spontaneously later. There was no tumor in the bladder.

bladder. The small calculus overlying the coccyx was interpreted as a calcification within a bladder tumor. Its midline location is probably due to displacement caused by the edema. There was swelling and edema in the region of the ureteral orifice cystoscopically and no evidence indicating a neoplasm. The stone could not be removed at this time, but was passed spontaneously two weeks later with complete recovery of all signs and symptoms.

FIG. 5. Deformity and irregularity of the bladder is present on the right. A calculus is visible in the intramural ureter (arrow). Note the obstruction of the right ureter with slight hydronephrosis and hydroureter. At cystoscopy marked swelling and edema of the right interureteric ridge was reported, and a smooth black calculus was visualized within the right ureteral orifice.

MacAlpine[5] has reported that by cystoscopic examination the region of the ureteral opening returns to normal in about one week after the passage of the stone. If this is true, the edema of the mucous membrane which remains for a time after pas-

sage of the calculus may be of importance in some of those patients with a fairly typical history of ureteral colic in whom the pain has subsided before the intravenous urogram could be done. The stone may have been passed and there may be no evidence of obstruction of the ureter, but the urogram may still show the typical defect of the bladder due to an intramural calculus. We have had occasion to interpret this finding as due to an intramural ureteral calculus which has recently passed. This has been helpful in establishing the cause of the abdominal pain.

SUMMARY

1. Calculi in the intramural portion of the ureter may produce sufficient edema and swelling of the bladder mucosa to be recognized as a filling defect in the bladder during intravenous urography.

2. Recognizable defects were present in 19 out of 26 cases of intramural calculus and thus are a common secondary sign of ureteral calculus in this location.

3. The more marked defects in the bladder due to a calculus may simulate the deformity caused by a carcinoma of the bladder.

4. Bladder deformity may be demonstrated for a short time following passage of the stone, which can be helpful in establishing a diagnosis in those patients who have passed the stone before the roentgen examination is done.

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DISCUSSION OF PAPERS BY DRS. ENGEL AND PETERSON AND HALL

DR. VINCENT J. O'CONOR, Chicago, Ill. Drs. Peterson and Hall have made an interesting and instructive contribution calling attention to the extreme deformity which often occurs on that side of the bladder wall in which an intramural calculus is impacted. The deformity seen in the cystogram is often much greater than the cystoscopic appearance would suggest. A note of warning must be sounded in regard to the interpretation of these deformities when they persist after the passage of a stone. Primary tumor of the lower end of the ureter may be confused with calculus. Since lime salt encrustation can and does occur on papillary tumors this differentiation may be troublesome.

We have seen 3 patients with tumors in lower end of ureter where particles of stone have passed but the edema and deformity persisted and fortunately we insisted upon a complete nephro-ureterectomy and cured the patient at an early stage of the disease.

(Slides were shown at this point.)

Dr. Engel is to be congratulated on emphasizing the great advantages which result from routine excretory urographic studies in patients with prostatic hypertrophy and other types of vesical neck obstruction. Wherever it is economically possible we make this a routine. Our interest is centered upon ruling out obstructive uropathy in the upper tract, stone, tumor and anomaly.

Since many men with enlarged prostates present themselves originally with a bladder full of clots we must always be sure that the patient is not bleeding from a renal or ureteral tumor or more commonly a bladder tumor.

Excretion urography is most important but is only an important part of a complete urological examination. Blood chemistry studies, urinary intake and output, renal function tests and cystoscopic study are equally important in what they may disclose of the patient's condition.

ROENTGEN DIAGNOSIS OF CHOLESTEATOMA OF THE MIDDLE EAR*†

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CHOLESTEATOMA is an epidermoid inclusion cyst which develops in the mastoid as a result of the extension of squamous epithelium into the middle ear through a perforation in the drum. It is associated with chronic purulent otitis media and may cause serious complications at practically any stage of the suppurative process. These complications include temporal lobe and cerebellar abscesses, thrombophlebitis of the sigmoid and lateral sinuses, purulent meningitis, purulent labyrinthitis and facial paralysis. Every cholesteatoma, due to the nature of its growth and its destruction of the surrounding structures, is a potentially dangerous disease. Chemotherapy and antibiotics have no effect upon its growth. Once a cholesteatoma is present, surgical procedures such as radical or modified radical operations should be considered. An epitympanic marginal perforation of the drum seems to be an almost absolute prerequisite in establishing the diagnosis of cholesteatoma, but the presence of such a perforation does not necessarily mean the presence of cholesteatoma. This seems to be definitely proved by accumulated histopathological data. A positive fistula test is one of the most important clinical signs of cholesteatoma if accompanied by the presence of an epitympanic marginal perforation of the drum or a bony defect of the lateral wall of the attic. Labyrinthine vertigo and deep-seated headaches are significant symptoms.

Although the above clinical signs should be paramount factors in the diagnosis of cholesteatoma of the middle ear, roentgen diagnosis should be attempted in every instance for three reasons: (1) to confirm the clinical diagnosis, (2) to detect early cases still lacking clinical symptoms, (3) to determine the extent of the cyst.

The difficulties of roentgen diagnosis of cholesteatoma of the middle ear are frequently observed. Areas of decreased density suggestive of cholesteatomas in roentgenograms many times fail to materialize as epidermal cysts at operation. Even more frequently, a cholesteatoma is discovered at operation in spite of negative roentgen findings. The number of cases belonging to the undiagnosed cholesteatoma group is larger than is apparent from statistics[3,6] because operation is too many times not performed because of inconclusive roentgen findings. Investigation of these diagnostic difficulties led to the present studies of the inherent limitations of roentgen diagnosis of cholesteatomata.

ANATOMY

It is natural that the first scope of these studies should concern itself with the establishment of the normal size of the mastoid antrum in the sclerotic bone. Entirely sclerotic temporal bones were used for measurements, because cholesteatomas almost invariably are found in non-pneumatized mastoids. Sixteen such specimens were studied in serial sections. None of them showed microscopic evidence of cholesteatoma. In three of the sixteen temporal bones there was an acute inflammation present in the middle ear. Chronic purulent otitis media with a central perforation of the drum was evident in one of the temporal bones. The width and the anteroposterior diameter of the mastoid antrum were measured under the microscope in every tenth consecutive section of

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† The study was assisted by the Hayden-Coakley Fund.

the bones which were serially cut in the horizontal direction. The height of the antrum was obtained by adding up the thickness of all the consecutive sections. In the four bones which were cut serially in the vertical direction only the height and the anteroposterior diameter could be measured. The width, the height and anteroposterior diameter refer to the corresponding diameters with the antrum in occipital position (Table 1).

Table 1

MEASUREMENTS OF MASTOID ANTRUM IN SERIAL SECTIONS

Serial Number	Greatest Width of Antrum in mm.	Smallest Width of Antrum in mm.	Average Width Near Base of Antrum in mm.	Height of Antrum in mm.	Greatest Antero-posterior Diameter of Antrum	Smallest Antero-posterior Diameter of Antrum	Proportion of All Consecutive Sections in Which Width is Greater than 6 mm.	Extent of Height of Antrum Along Which Width is Greater than 6 mm.	Nature of Surrounding Bone	Middle Ear	Audiogram	Age	Section: V—vertical H—horizontal
1	10.5	2	3–4	10	6.5	3	1/2	5.5	Sclerotic	Normal	Normal	41	H
2	—	—	—	11	10	4	—	—	Sclerotic	Normal	Normal	41	H
3	11	3.5	3.5	6.2	7	2	4/5	5	Sclerotic	Normal	—	45	H
4	11.5	5	5	12	7	4	2/3	7.5	Sclerotic few cells	Acute purulent otitis media	—	45	H
5	9	—	7	—	—	—	—	—	Sclerotic	Acute purulent otitis media	—	47	H
6	8	6	6	8	6.5	4.5	1/2	3.5	Diploic	Acute purulent otitis media	—	47	H
7	9.5	3	5	9	9	3	1/3	3	Sclerotic	Normal	—	52	H
8	10.5	3.5	5	10	8	3.5	2/3	6.5	Diploic	Normal	—	52	H
9	10.5	7	7	9	8	4	3/4	7.5	Diploic	Normal	—	54	H
10	10	4	5	8.8	7	5	2/3	5.3	Sclerotic	Normal	—	54	H
11	9	5	5	11.5	8	4	1/4	3.3	Sclerotic	Normal	Good	55	H
12	9	4	5	5.5	5	2	1/2	2.8	Sclerotic Diploic	Normal	Normal	70	H
13	—	—	—	9	10	7	—	—	Sclerotic	Normal	—	35	V
14	—	—	—	4	—	—	—	—	Sclerotic	Chronic purulent otitis media	Normal	52	V
15	—	—	—	7	—	—	—	—	Sclerotic	Normal	—	65	V
16	—	—	—	7	—	—	—	—	Sclerotic	Normal	—	75	V

Going upward from the base towards the roof of the antrum, the width showed considerable variation at different levels. Invariably the smallest figures were found close to the base. The width of the antrum grew gradually or abruptly at the level of the lateral semicircular canal. The difference between the narrowest and widest figures were not found at all levels along the vertical axis of the antrum. Varying from one bone to the other one-third to four-fifths of all consecutive sections showed the mastoid antrum wider than 6 mm. A separate column of the table indicates what proportion of all consecutive sections measures more than 6 mm.

The width of the antrum in its inferior third was less than 6 mm. in all but two bones.

The vertical diameter (height) of the antrum proved to be greater than 10 mm. in three of the sixteen bones of this series. The height ranged from 4 mm. to 12 mm. in different temporal bones.

The anteroposterior diameter of the antrum showed equally great variations. It measured from 2 mm. to 10 mm. at various levels along the vertical axis. The variation in the anteroposterior diameter in the same antrum ranged from 2 mm. to 6 mm. As a rule the width of the antrum was larger than its anteroposterior diameter in each individual section. It was found that the thickness of the bony walls of the antrum was usually but not invariably in inverse relationship to the anteroposterior diameter of the same. The inner table which separates the antrum from the cerebellar fossa may be as thin as 0.75 mm. or may measure 4–6 mm. in thickness.

There was nothing characteristic about the shape of the mastoid antrum which would allow a comparison with pathological changes. Its form was somewhat irregular in most of the bones. In some it was ovoid, in others circular. In all bones, however, there was a progressive decrease in the width of the antrum going from the tegmen towards the base. It has a roughly conical shape with a blunt tip pointing downwards.

The bone which surrounded the antrum was mostly sclerotic. In a few instances diploic bone formed the wall in some places and occasionally a few very small cells containing thick wall were found adjacent to the antrum.

An attempt to reach conclusions regarding the standard size and shape of the attic proved to be fruitless. Variations in all three diameters, width, height and anteroposterior, were so prevalent that useful conclusions could not be made. The thickness of the lateral wall of the attic and tegmen tympani equally shows great variations adding to the difficulties of roentgen diagnosis of this cavity. Only extensive bone destruction in the attic can be interpreted as being pathological.

PATHOLOGY

Cholesteatoma may develop in the antrum, in the attic or may invade both of these cavities. On reviewing 50 consecutive radical mastoid operations at which cholesteatoma was found, the pathologic condition was localized to the mastoid antrum in 30, to the attic alone in 10, and to both antrum and attic in 10 cases. Some of the cholesteatomas seen at operation were small, others were very large occupying a considerable portion of the mastoid and petrous bones and extending through the posterior wall of the external auditory canal. Even a small cholesteatoma can, however, destroy the walls of a small antrum or attic and cause serious complications. This is especially true for the attic.

The height of the antrum measured only 4–7 mm. in four of the sixteen bones studied in serial sections. This would mean that in such bones, 6–8 mm. of the surrounding bony walls would have to be destroyed before the roentgenogram would appear indicative of cholesteatoma. That much destruction would probably already have led to intracranial encroachment although measurements in the films would indicate a normal sized antrum.

The rate of growth of a cholesteatoma is another unknown factor. Usually the enlargement of the antrum due to the bone destruction is a very slow process. Repeated roentgen studies of the same patient with intervals of eight to ten years reveal an enlargement of only 2–3 mm., sometimes more. Sudden flare-ups followed by rapid bone destruction are occasionally observed by the otologist.

CRITERIA FOR ROENTGEN DIAGNOSIS OF CHOLESTEATOMA

In the opinion of most roentgenologists, an enlargement of the mastoid antrum is the most important and indispensable feature in the roentgen diagnosis of cholesteatoma of the middle ear. According to MacMillan[3] the normal sized antrum is 6 mm. wide by 10 mm. high in a sclerosed mastoid. Any increase in size of the antrum

is considered to be due to erosion of the bone by growing cholesteatoma. A condensation of the periantral bone is considered as additional evidence.

The intimate spatial relationship of the antrum and attic to the very dense labyrinthine mass greatly reduces the usefulness of certain projections in the diagnosis of cholesteatoma of the middle ear. On the other hand, a certain degree of uncertainty in identifying the different structures on the films exists many times due to the complicated interrelationships of the bony structures in the mastoid and petrous bone areas. In order to eliminate both these sources of error, dry skulls were sawed through a sagittal plane so that the attic and antrum were halved in the anteroposterior diameter without loss of their bony walls.

Antrum. Radiopaque material was first put into the antrum completely filling the entire cavity. Roentgen studies of these skulls showed that most of the antrum is superimposed upon the dense labyrinthine mass in Law's position (Fig. 1, *A* and *B*). In both the Stenvers and occipital (MacMillan[3]) positions the entire antrum is projected laterally from the labyrinth. The occipital position (Fig. 2*B* and 4*B*) has definite advantages in demonstrating the antrum because this cavity is almost never crossed by bony shadows of the base of the skull in contrast to the Stenvers position. In Mayer's position the antrum is projected into the mastoid process and the labyrinthine mass does not overlap the antrum (Fig. 3*B*). Measurement of the radiopaque filling in the skull and on the films showed negligible discrepancy within a range of 1 mm. as to the height of the antrum. The measurement of the width of the antrum did not show any variation on the roentgenogram as compared with skull

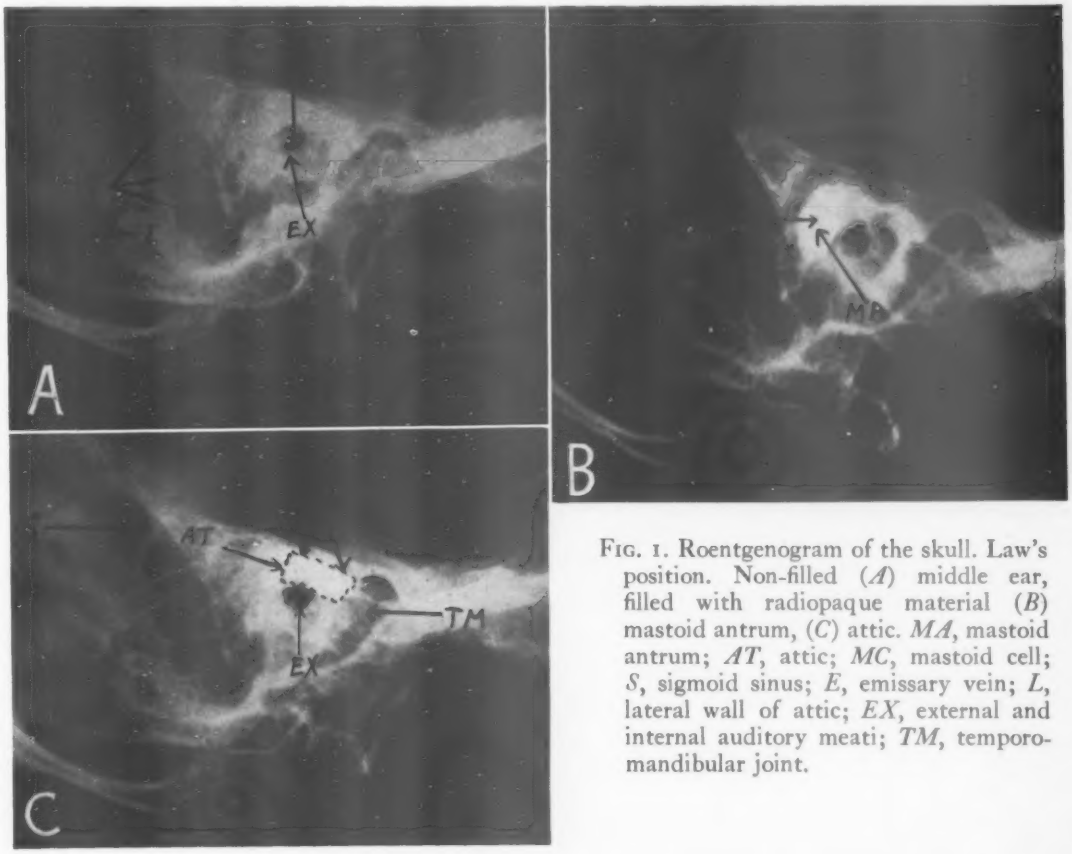

Fig. 1. Roentgenogram of the skull. Law's position. Non-filled (*A*) middle ear, filled with radiopaque material (*B*) mastoid antrum, (*C*) attic. *MA*, mastoid antrum; *AT*, attic; *MC*, mastoid cell; *S*, sigmoid sinus; *E*, emissary vein; *L*, lateral wall of attic; *EX*, external and internal auditory meati; *TM*, temporomandibular joint.

measurements. This finding is somewhat surprising in view of the fact that due to the position of the skull, angulation of the roentgen tubes, and anatomical variations, a certain amount of distortion was expected.

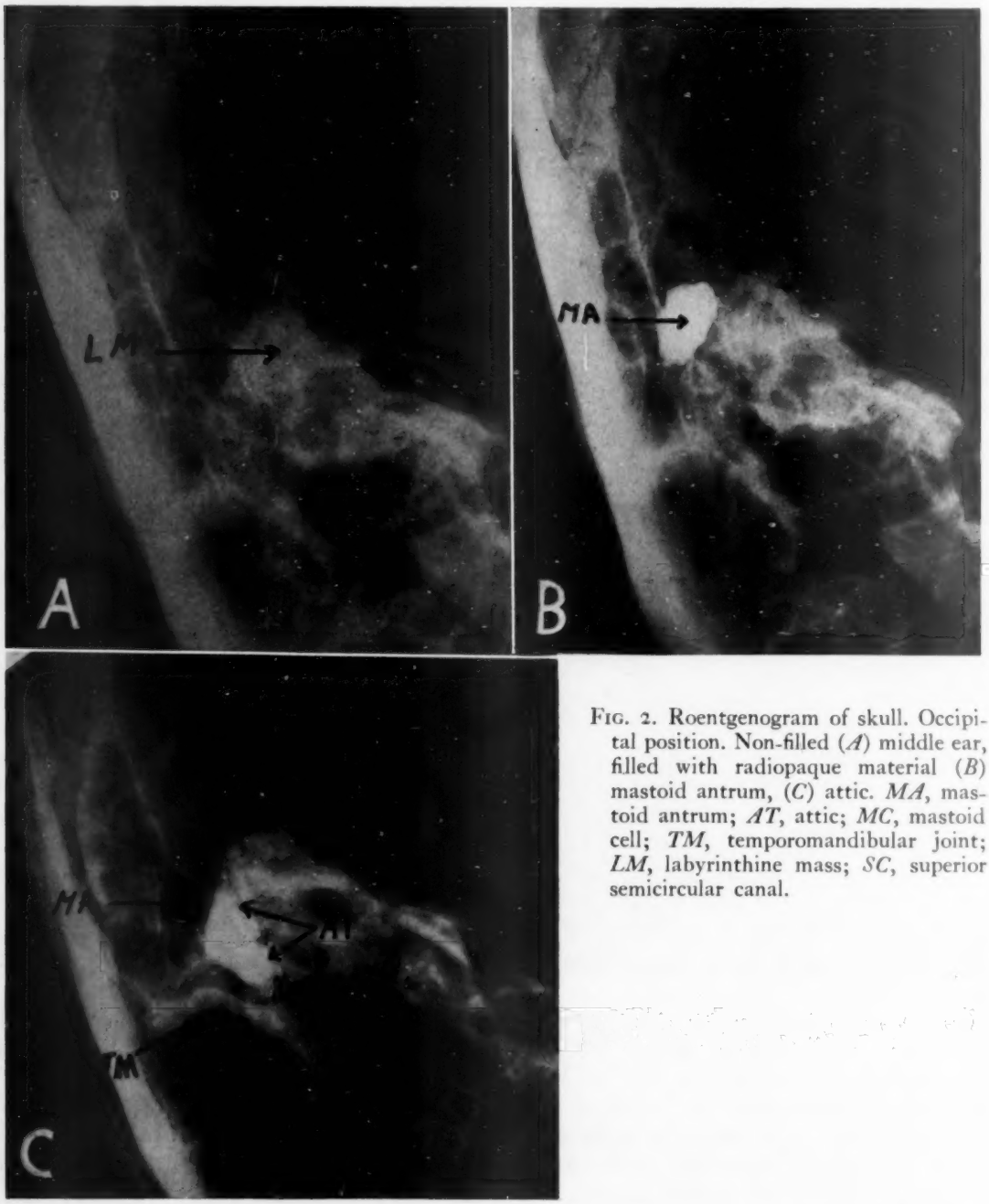

Fig. 2. Roentgenogram of skull. Occipital position. Non-filled (*A*) middle ear, filled with radiopaque material (*B*) mastoid antrum, (*C*) attic. *MA*, mastoid antrum; *AT*, attic; *MC*, mastoid cell; *TM*, temporomandibular joint; *LM*, labyrinthine mass; *SC*, superior semicircular canal.

Attic. After removal of the radiopaque material from the antrum the attic was filled in the same way. Both in Law's and Stenvers' positions almost the entire attic falls into the dense shadow of the labyrinthine mass (Fig. 1*C*) and the lateral wall of the attic. In the occipital position most of the attic is projected below the dense labyrinthine shadow (Fig. 2*C*). In Mayer's position the labyrinth is clearly shifted

away from the attic. However, with the exception of a small portion of the attic which is projected into the tympanic cavity and the external auditory canal, the attic is covered by the lateral wall of the attic and the posterior wall of the external auditory canal (Fig. 3*C* and *D*, and 4*D*).

OBSERVATIONS

Roentgen studies of the mastoid were reviewed in 70 consecutive unselected cases in which a radical mastoid operation was performed. In 25 of the 70 cases, the operative findings did not confirm the roentgen diagnosis. In 17 patients cholesteatoma was found at operation while roentgen studies failed to demonstrate its presence. Out of the 17 cases, the cholesteatoma was located in the attic in 7, and in the mastoid antrum in 10. All of the attic cholesteatomas remained undetected in roentgen studies. Cholesteatomas of the mastoid antrum fared much better; of 34 cases, only 10 were not demonstrated. On the other hand, roentgenograms were thought to give the characteristic picture of cholesteatoma in 8 cases in which operation failed to reveal epidermoid pathology of macroscopic or microscopic nature.

Using enlargement of the mastoid antrum as evidence of cholesteatoma resulted in erroneous conclusions in about one-third of the cases of this series. The frequency of failure to demonstrate a cholesteatoma by roentgenograms greatly outnumbers those instances in which operation fails to reveal a cholesteatoma when the films show an apparently enlarged antrum. Routine microscopic studies helped to reduce the number of diagnostic errors in cases where gross evidence of cholesteatoma was not found at operation. Complete spontaneous liquefaction and disappearance of the cholesteatoma before operation, even though the cavity does not show macroscopic evidence of it at operation, is not likely to occur.

Law's position, occipital position (MacMillan) and sometimes Stenvers' position were used as standard techniques in the above series. MacMillan's rule was followed as to the normal size of the mastoid antrum in a sclerosed mastoid.

COMMENT

Measurements of the mastoid antrum under the microscope necessitate certain changes as to the evidence which is generally accepted as indicative of pathological enlargement of the mastoid antrum in roentgenograms. In a considerable number of all consecutive sections of each temporal bone studied in this series, the width proved to be greater than 6 mm., which is accepted as being the upper limit of normal. Only for the inferior third of the antrum in the occipital position could 6 mm. be the limit of normal variations. Near the base of the antrum the width rarely exceeds 6 mm. As long as enlargement of the antrum is the only reliable basis for roentgen diagnosis of a cholesteatoma, our attention should be concentrated on the width of the inferior third of the antrum.

Measurements of the height of the antrum in sclerosed temporal bones showed that 11 mm. rather than 10 mm. should be considered as the upper limit of normal.

The two changes described above may help to eliminate some of the roentgenological errors leading to unnecessary operations. Radical mastoid operation, however, is essentially a prophylactic operation which is being performed in order to forestall serious complications. The otologist is justly concerned in making an early diagnosis. Most of the diagnostic errors in roentgen studies occur in early cases. Early diagnosis is impossible in the vast majority of cases while roentgen diagnosis in late cases (when the cholesteatoma has reached a considerable size) is likewise impossible in a large number of cases. One of the reasons for these failures lies in the anatomical variations both in the antrum itself and in the surrounding bony structures. It may take many years before a cholesteatoma developing in a 4 by 5 mm. antrum can be demonstrated in roentgenograms. It is obvious that a so-called pathological en-

largement of the antrum by a cholesteatoma can be demonstrated in roentgenograms much earlier if the antrum was large to begin with and surrounded by thin bone. The danger of intracranial complication, however, may be the same—being independent of the original size of the antrum.

The anteroposterior diameter of the antrum shows great variations not only from section to section but in individual sections. Due to the irregular shape of the cavity the antrum may be several millimeters shallower laterally than medially, or vice versa. A thin layer of air will be present in the shallow portion making roentgen demonstration impossible. In addition, in a significant number of cases the antrum is separated from the endocranium by a thick cortical bone. An air or cholesteatoma containing cavity located deep in the center of a large sclerotic bone does not lend itself to roentgen demonstration.

Variations in thickness of the lateral wall of the attic make demonstration of the attic quite uncertain. A 7 mm. thick sclerotic lateral wall may make roentgen demonstration of the air or cholesteatoma containing cavity of the attic impossible. On the other hand, the great variation in the size and shape of the attic in normals makes it impossible to establish normal useful values for measurement.

Tomographic examination may be helpful in visualizing cavities surrounded by thick layers of sclerotic bone.[5]

Limitations inherent in different positions are another factor that makes roentgen evidence of secondary importance in the diagnosis of cholesteatoma. Law's position does not, as a rule, give valuable information because most of the antrum and of the attic is overlapped by the dense labyrinthine mass.[8] In addition, the lateral wall of the attic is superimposed upon the attics in this position (Fig. 1*C*). Large cholesteatomas extending into the sinodural angle, destroying the anterior margin of the sigmoid sinus, or breaking through the roof of the antrum and attic are exceptions to this rule. In order to demonstrate a defect in the roof of the attic, the roentgen rays must hit the defect tangentially. Therefore a number of films taken at slightly different angles may be necessary in order to demonstrate the defect. An artificially made bony defect measuring 3 by 3 mm. could not be demonstrated in the dry skull in repeated roentgenograms. Demonstration of such pathology amounts actually to luck.

The Stenvers position demonstrated the antrum because it is overlapped only by the mastoid bone. However, the base of the skull frequently obscures visualization of the antrum in this position. A mastoid containing small cells with thick bony septa may make it impossible to obtain clear outlines of the antrum. This statement applies to all positions. Stenvers' position is of little help in the diagnosis of a cholesteatoma which is localized to the attic because this cavity is superimposed by the shadow of the labyrinthine mass.

The occipital position is more satisfactory in demonstrating the antrum (Fig. 2*B* and 4*B*), as compared with Stenvers' position. The base of the skull is rarely superimposed upon the antrum in this position. Demonstration of the attic in the occipital position is rarely possible in a satisfactory way. It was found that in roentgenograms of dried skulls taken in the occipital position, no definite outline of the attic was discernible. The exact outline of the attic was verified in the same skulls in the same roentgen position after filling the attic with radiopaque material. Roentgen studies of dried skulls in the occipital position showed that the shape and size of the antrum could not be invariably demonstrated, though a good sized antrum was visualized if the cavity was filled with radiopaque substance in the same temporal bone.

Mayer's position[4] or its modifications satisfactorily demonstrate the antrum because the labyrinth is projected towards the tip of the petrous bone. The usefulness of this position, however, is a very limited one in demonstrating the shape and size of the attic.[1,7,9] A dense sclerotic lateral attic wall

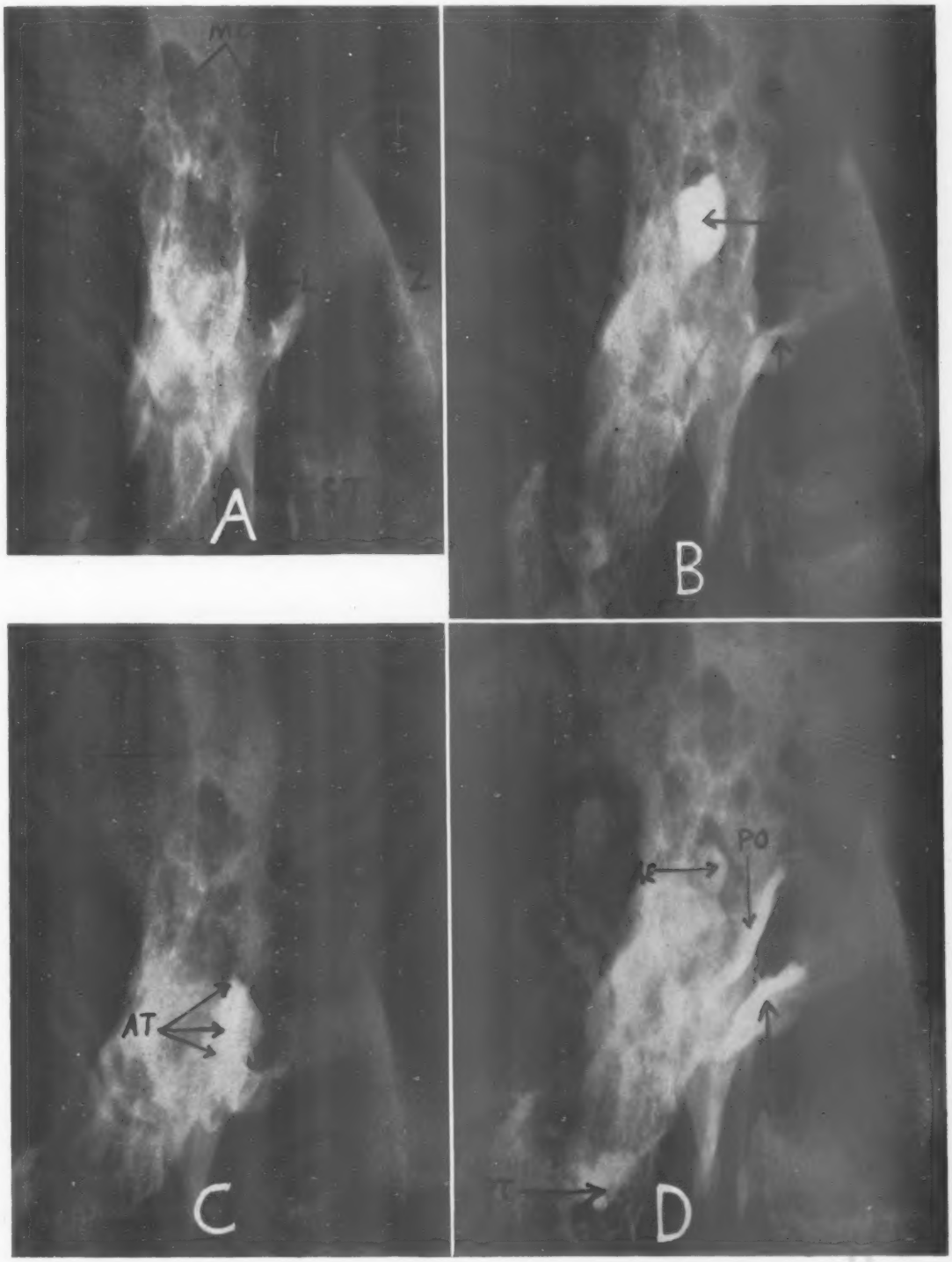

Fig. 3. Roentgenogram of skull. Mayer's position. Non-filled (*A*) middle ear, filled with radiopaque material (*B*) mastoid antrum, (*C*) attic. Anterior and posterior wall (*D*) of the bony external auditory canal coated with radiopaque material which reaches tympanic ring. Radiopaque marker on arcuate eminence and petrous tip. Note shape and size of attic cannot be demonstrated without filling. *ANT*, anterior wall of external canal; *PO*, posterior wall of external canal; *MA*, mastoid antrum; *AT*, attic; *L*, lateral wall of attic; *E*, emissary vein; *S*, sigmoid sinus; *MC*, mastoid cell; *Z*, zygoma; *PT*, petrous tip; *AE*, arcuate eminence; *ST*, styloid process.

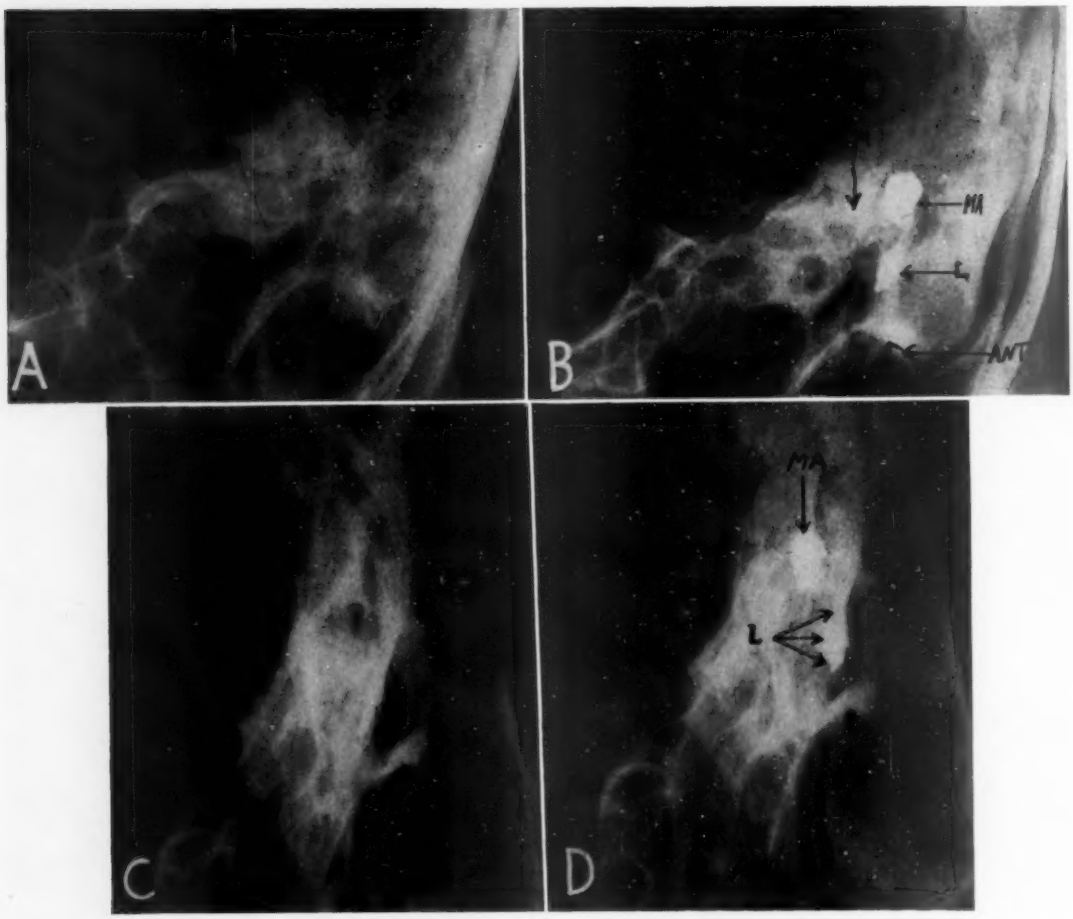

Fig. 4. Roentgenogram of skull. Non-filled (*A*) middle ear in occipital position. Mastoid antrum filled (*B*) and inner side of lateral wall of attic coated with radiopaque material in base position. Same skull without filling (*C*) in Mayer's position. Same skull in Mayer's position (*D*) with same filling as in (*B*). *MA*, mastoid antrum; *L*, lateral wall of attic; *LM*, labyrinthine mass; *SC*, superior semicircular canal; *ANT*, anterior wall of external auditory canal.

and a similar posterior wall of the external auditory canal may completely cover the major portion of the attic (Fig. 3*C* and *D*, and 4*C* and *D*). Therefore in Mayer's position the only acceptable evidence for a cholesteatoma of the attic is an extensive total destruction of the lateral wall of the attic or posterior wall of the external auditory canal. On the other hand, a large bony defect of the lateral attic wall is easily detected by otoscopic examination. In the presence of a thick lateral attic wall, a cholesteatoma of the attic may remain undetected in roentgen studies, no matter what position is used. The dura may be pathologically exposed or a temporal lobe abscess may already be present and the roentgenograms still appear negative. Many of these complications show a notoriously symptom-poor course and both the patient and surgeon become overconfident about the potential danger, particularly in the presence of a negative roentgen report. If we keep in mind that not all otogenic intracranial complications can be cured and that a purulent labyrinthitis will invariably lead to total and irreversible deafness, we have to realize the paramount importance of the clinical examination. Especially is this true for cholesteatomas of the attic.

Out of 100 cases of cholesteatoma of the middle ear about 20 are localized to the attic. In most of them even the best roentgen studies will be of very limited help and negative roentgen findings should not be a deciding factor in the treatment of such cases.

Cholesteatoma very rarely develops in a pneumatized mastoid bone. In spite of still existing controversy on this issue, proved cases of cholesteatoma in a pneumatized mastoid bone are occasionally observed at operation. Sclerotic mastoid is the rule, however, in cholesteatomas.

"Cholesteatoma is present in practically all cases of chronic mastoiditis according to many otologists"[2] is a statement which cannot be confirmed in view of our histopathological data. A great number of sclerotic bones have been observed with a history of long standing suppuration in which serial sections failed to reveal any evidence of a cholesteatoma. To this group belong practically all cases in which the perforation of the drum does not extend to the epitympanic region and to the tympanic ring. The vast majority of the patients belonging to the latter group do not constitute a surgical problem and hardly any help in the management of these cases can be expected from roentgen studies. This is an important reason why a roentgen diagnosis of chronic mastoiditis should be used with great reserve.

SUMMARY

1. Measurements of the mastoid antrum of unpneumatized bones in serial sections showed that the normal antrum is wider than 6 mm. in its upper two-thirds but almost never in its lower third near the base. The upper limit of the normal height is 11 mm. with occasional exceptions up to 12 mm. Considerably smaller mastoid antra, however, occur in normal unpneumatized bones. The standard shape and size of the attic could not be established because of the numerous anatomical variations of this cavity.

2. Cholesteatomas of the middle ear cannot be diagnosed by roentgen studies if one or more of the following conditions exist: (a) cholesteatoma fills the mastoid antrum but does not destroy its walls, (b) cholesteatoma occurs in a deeply located antrum surrounded by thick inner table and cortical bone, especially if the anteroposterior diameter is small (flat antrum), (c) cholesteatoma is present in a small antrum, (d) cholesteatoma is localized to the attic—very large cholesteatomas are exceptions under this rule, (e) cholesteatoma develops in a mastoid on which a simple or a radical operation was previously performed.

3. The only roentgen evidence of an attic cholesteatoma is destruction of the lateral wall of the attic, which, if extensive enough, can be detected in Mayer's position, or a bony defect in the roof of the attic which is occasionally demonstrated in Law's position. Both of these lesions represent a late stage of the disease.

4. Demonstration of the attic on roentgenograms is not possible in the projections known, including Mayer's position.

5. Roentgen studies of cholesteatoma of the middle ear lead to erroneous conclusions in about one-third of all operated cases. The vast majority of roentgen diagnostic errors occurs in cases in which cholesteatoma is found at operation while preoperative roentgen studies failed to demonstrate their presence. All cases of attic cholesteatoma remained undetected in this series. In an average of twenty out of one hundred cases, the cholesteatoma is localized in the attic.

6. An enlarged antrum in roentgenograms indicates cholesteatoma only if it is associated with an epitympanic, marginal perforation of the drum. If there is a contradiction between clinical findings and the roentgen studies, the treatment of the case should be directed by the clinical evidence.

7. Close cooperation between the otologist and roentgenologist is essential in reaching useful conclusions and will result in improved diagnostic procedures.

I wish to acknowledge with much appreciation

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MALIGNANT DEGENERATION OF BENIGN GIANT CELL TUMOR OF BONE*

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RECENTLY an increasing number of articles have appeared in the literature considering irradiation with roentgen rays and radium as hazardous in the treatment of benign giant cell tumor of bone. It is admitted by a large majority of the investigators that radiation therapy in most instances leads to satisfactory anatomic and functional results and that the outlook for a permanent cure is good. However, cases are being continually observed in which after many years malignant osteogenic sarcoma arises at the site of the original benign giant cell tumor, often with fatal results. There is now a tendency to attribute to the roentgen rays a certain contributory role in such a development and therefore an effort is being made to re-evaluate the therapeutic approach in non-malignant tumors of the bone.

It is the purpose of the present article to study the various possibilities leading to a malignant degeneration of benign giant cell tumors of the bone and to analyze the relative incidence of such degeneration following different methods of treatment. In doing so, perhaps there is a certain advantage in first discussing the possibility of malignant degeneration due to irradiation. Here, a sharp distinction must be made between the cases which were grossly over-irradiated and those in which the customary doses and technique were used. Then, to gain a comparative evaluation, the possibility of a spontaneous malignant degeneration is considered. Finally, the rather voluminous literature dealing with the occurrence of sarcomatous changes following various surgical procedures or other methods of treatment is reviewed. To this a series of personal cases is added and the significance of the several possible causative factors is discussed.

MATERIAL

The study is based on a series of 77 cases of benign giant cell tumor of bone, treated at Harper Hospital, Detroit, from 1923 to 1947 inclusive. The method of treatment consisted of curettage and postoperative roentgen therapy from 1923 to 1929 and of roentgen therapy as the principal procedure since 1930. The detailed technique of the roentgen therapy was described in former publications.[9,18]

In the entire group, 5 of the cases are known to be dead, 3 from incidental disease and 2 as a result of subsequent sarcomatous change of the benign giant cell tumor. Five cases are untraced, 1 of which had a malignant degeneration of the benign giant cell tumor. In an additional 4 cases a sarcomatous change of the benign giant cell tumor developed, but after further treatment, either by surgery or irradiation, a satisfactory final result was obtained.

Thus, the number of the cases showing malignant degeneration was 7 out of 77, or approximately 9 per cent. All of the cases are presented in detail. In 4 of them the malignant degeneration occurred following routine roentgen therapy, in 1 it apparently was spontaneous and in 2 it followed surgical procedures. An eighth case is included, in which fibrosarcoma of the sternum developed eighteen years after gross over-irradiation for thymic enlargement.

The Development of Sarcoma in Grossly Over-irradiated Bone. Any tissue that has been grossly over-irradiated undergoes degenerative changes which after many

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years may culminate in malignant neoplasia. If the effect was superficial the changes appear primarily in the epithelium of the skin, leading to marked atrophy followed later by ulceration, secondary infection and eventually by the development of carcinoma. If the action occurred in the deeper layers of the body, the late sequelae may consist of degenerative processes of the connective tissue elements resulting in sarcoma of the soft parts or bone. A study of the relative incidence shows that with roentgen rays, especially of the lower voltage variety, the carcinoma is very much more common than the sarcoma. In 1940, 28 cases of malignant roentgen neoplasms which followed excessive doses of roentgen rays were reviewed at Harper Hospital.[31] Of these 25 represented carcinomas and only 3 were sarcomas, 2 having been classified as being of subcutaneous origin, and 1 mixed, of subcutaneous and osseous origin. However, when radium, or any other member of the naturally radioactive families, is absorbed internally, as was the case with the ingestion of the luminous dye in the watch dial painters or with the drinking of radioactive waters, an irreversible deposition occurs in the bone, leading to the formation of osteosarcoma in a large majority of the cases. The same situation arises when a radium salt solution is injected into a joint or into the soft tissues lying close to the bone.

A study of the group of cases observed at Harper Hospital revealed that it made little difference whether the over-irradiation was accomplished by the administration of a single large dose or of repeated smaller doses, or by the continuous action over a long period of time of minimal doses, as is the case with radium. If the tissue changes once became permanent, a subsequent malignant degeneration at any later time could not be precluded, despite the complete discontinuation of the irradiation or any other precautionary measure. The following case of roentgen sarcoma illustrates this point.

CASE 1. H. B., white female, was first seen at Harper Hospital on January 2, 1936, at the age of twenty-one years.

The patient gave a history of having received roentgen treatment over the upper chest when she was three years old, apparently for enlargement of the thymic gland. The physician who administered the treatment has died since then and therefore the factors of the irradiation could not be secured.

A rather marked telangiectasis and atrophy of the skin of the treated area developed early in life. At the age of seventeen the skin started to break down and the patient had two minor operations for removal of ulcers and cornifications.

When examined at Harper Hospital on January 2, 1936, there was a large area of telangiectasis of the upper chest and lower neck anteriorly. Throughout this area there were several cornified excrescences, giving the impression of hyperkeratoses and near the right clavicle there was a larger tumor suggestive of malignant degeneration.

From January 2 to March 31, 1936, the patient was hospitalized and several plastic operations with skin grafts were done.

The microscopic examinations of several specimens of the skin revealed changes incident to over-irradiation. The report on the tumor was that of a fibrosarcoma of rather active growth capacity (Fig. 1).

By October, 1936, the patient developed several new tumors, chiefly at the borders of the grafts, and at this time there was a beginning involvement of the sternum. Since further operation was out of the question, supervoltage roentgen therapy was started, despite the fact that the patient already had severe irradiation injuries.

Several series of treatments were given with rather large total doses. The sarcoma, however, continued to progress. On September 13, 1937, there was already a large fungating mass arising from the sternum and there was evidence of metastases to the right supraclavicular and lower cervical lymph nodes (Fig. 2).

The patient received additional roentgen therapy until February, 1938, when it was discovered that she was two months pregnant. On April 11, 1938, she was given 104 mg. of colloidal lead phosphate intravenously, but no further irradiation. The impression was gained that the pregnancy did not substantially influence the rate of growth of the sarcoma. On

June 15, 1938, the patient moved to another state.

Information was gained through a friend of the patient that she died in August, 1938, the outcome of the pregnancy being unknown.

In the light of this observation it may be of interest to review briefly the literature giving examples of osteosarcoma produced by exposure to roentgen rays or radium during the course of treatment of lesions of some other structure than the bone. Hatcher[14] and Cahan and his coworkers[3] collected 24 cases of osteosarcoma which followed exposure to irradiation. Of these, 17 resulted from roentgen therapy for tuberculous arthritis, 1 from similar treatment for acute arthritis, and 6 from radium treatment. The 18 cases treated with roentgen rays were published

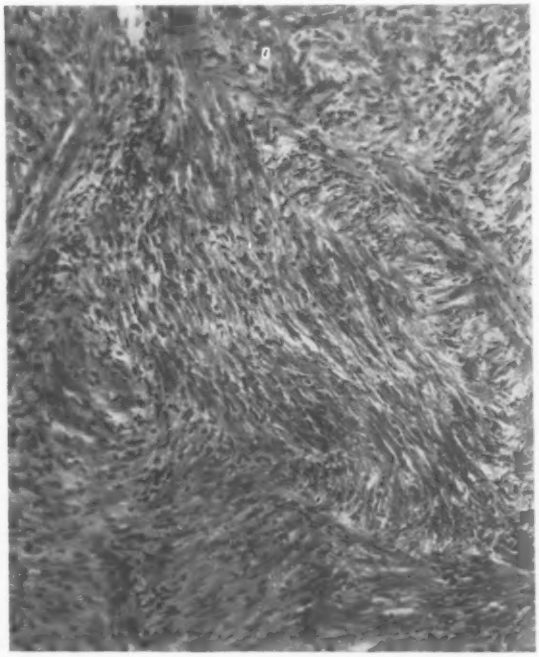

Fig. 1. Case 1. Fibrosarcoma, photomicrograph of lesion shown in Figure 2.

by 11 authors, all in the European literature, between 1922 and 1937. The given dose is not specified in the majority of instances, but the description of the sequelae indicates that they were large and extended over long periods. The 6 cases exposed to radium were reported by Martland[21] and Nørgaard.[24]

Martland,[21] in 1931, reviewed the data pertaining to the damage caused by radium in the watch dial painters. Of the 18 per-

Fig. 2. Case 1. Fibrosarcoma of the soft tissues and of the sternum in a grossly over-irradiated area, eighteen years after roentgen therapy for thymic enlargement.

sons who died, 5 had bone sarcomas, 1 in the femur, 1 in the scapula, 1 in the pelvis, 1 in the pelvis and femur, and 1 in the pelvis and orbit. There were 3 additional cases of osteosarcoma who were still alive. It is stressed by Martland that, in addition to the various known forms of blood dyscrasias, severe "radiation osteitis" with pronounced deformities of the bones constituted the outstanding feature in the final stages of the disease.

Some very interesting experiments were carried out in relation to Martland's observations by Uehlinger and Schürch.[30] These authors placed a mixture of either mesothorium or radium (0.002–0.005 mg.) and vaseline (0.2 gm.) in the marrow cavity of the femur of rabbits and were able to produce osteosarcoma in 57 to 63 per cent of the animals within eighteen months. They also briefly describe a case in

which heavy doses of radium and roentgen rays given fourteen to eighteen years previously for a chromophile adenoma of the pituitary gland resulted in a sarcoma of the ethmoid bone.

In 1939, Nørgaard[24] reported a case which received treatment for chronic arthritis at the radium springs of St. Joachimsthal in Bohemia. The patient had radium baths in 1928, 1929, 1931, 1934 and 1937. In 1928 she also had an injection of radium chloride into the right shoulder joint and in 1929 two more injections into the right knee joint. A radium necrosis developed in both joints, followed eight years after the injections by a "spindle cell sarcoma and fibrosarcoma in chronic periostitis" of the proximal end of the right tibia. There are other similar cases described in the literature in which sarcoma of the bone eventually resulted from the prolonged effect of radium deposits in the skeleton. In all instances radioactivity of the osseous tissue was demonstrated at the time of the discovery of the tumor. Gettler,[12] after examining the bones of a dial painter, exhumed five years after death for litigation purposes, found that the radium was still present throughout the entire skeleton.

Hatcher[14] reported 3 cases in which sarcoma had developed in bone which had been in the field of irradiation directed toward an independent lesion. In the first case, the original lesion represented a "benign chondroblastoma" of the proximal epiphysis of the tibia which was curetted and treated by intensive roentgen therapy. Four years later a sarcoma of the proximal end of the fibula appeared. The skin was pigmented and atrophic with telangiectasis but there was no ulceration. An amputation was done. Soon, metastases to the lungs developed and the patient died two years later. In the second case, the primary lesion was a benign giant cell tumor of the radial side of the right wrist. Following biopsy three radium applications were made and several series of roentgen therapy were given. Ulceration, infection and bone necrosis developed, necessitating osteotomy of the ulna to correct deformity. Eleven years later "fibrosarcoma with tumor cartilage" of the distal end of the ulna developed. Ten years after the excision of this second tumor there was no evidence of local recurrence or metastasis. In the third case a chondrosarcoma developed in the seventh rib eleven years after roentgen therapy of a mammary carcinoma. Here no appreciable skin changes due to irradiation were present.

Malignant Degeneration of Benign Giant Cell Tumor Following Irradiation with Ordinary Doses. It is not the intention to review here the rather long series of articles dealing with the various phases of radiation therapy or the results obtained from such treatment. However, some noteworthy information may be gained from a consideration of those instances in which either individual cases or smaller groups of cases are reported as representing samples of malignant degeneration of a benign giant cell tumor incident to or resulting from the irradiation.

In the early era of radiation therapy no attention was paid to the possibility of this type of malignant degeneration. Therefore the first investigators concerned themselves mostly with a study of the value of the radiation therapy as compared to that of the surgical methods used at that time.

Later, when the results were compiled statistically, it gradually became apparent that some of the cases irradiated died subsequently with sarcomatous manifestations.

Peirce,[25] in 1932 and again in association with Lampe[26] four years later, reported a series of 40 cases treated by various methods at the University of Michigan Hospital in Ann Arbor. In 15 cases treated by irradiation alone there were no instances of malignancy, but of 8 cases treated by a combination of surgery and roentgen therapy 2 had osteogenic sarcoma. A closer scrutiny of these 2 cases, however, shows that both the surgical intervention and the irradiation were of only incidental signifi-

cance and that the divergent morphology of the tumors was the important factor. In one case the histopathologic study indicated a "chondro-osteogenic sarcoma arising in giant cell tumor" and in the other a "giant cell tumor with high degree of cellularity." This latter case at the time of the microscopic examination already had metastases to the lungs.

Francisco, Pusitz and Gerundo,[10] in 1936, published the case history of a girl, nine years of age, in whom a bone cyst treated by irradiation turned into sarcoma about eight years later. At the age of nine months the patient fell and hurt her right leg. A roentgen study was made elsewhere and on the basis that the condition represented sarcoma roentgen therapy was given for a year, the factors being unspecified. The patient became worse and at this time was referred to Dr. Francisco. Roentgenograms showed a "soap bubble" central lesion in the upper end of the tibial shaft which was interpreted as a bone cyst. The leg was immobilized with cast and brace until the age of four and one-half years, when the lesion was regarded as cured. At the age of nine years the pain returned after trauma to the site of the original lesion and subsequent roentgenograms revealed an appearance of the upper half of the tibia which was diagnosed as a low grade osteomyelitis due to roentgen exposure but which on biopsy proved to be a chondrosarcoma. An amputation was done.

Doub, Hartman and Mitchell,[7] in 1938, in studying the results in 22 cases of giant cell tumors of the Bone Tumor Registry in which radiation therapy was used found that a malignant degeneration occurred later in 3 cases. In 2 cases a combination of curettage and roentgen therapy, and in the third case roentgen therapy alone constituted the treatment. These authors also described a case of their own, originally confirmed as a benign giant cell tumor by biopsy, which within seven months of the time roentgen therapy was started with small doses developed into fibrosarcoma. The question was raised whether small repeated doses of roentgen rays may not have a stimulative effect leading, especially in association with traumatizing surgical procedures, to malignant transformation. In the same year Brunschwig[2] reported the late results in 9 cases of benign giant cell tumor which he observed periodically for five years or more after treatment. In 8 of the cases curettage followed by roentgen therapy constituted the method of treatment. The ninth case, which is described in detail, had a giant cell tumor of the lower end of the radius, which in 1921 was "scraped" and in 1922–1923 received numerous roentgen exposures and radium application, the factors being unknown. A draining sinus developed with bone necrosis, for which later sequestrectomy was performed. In 1929 the radial angulation of the hand was corrected by osteotomy above the lower end of the ulna. In 1934 a tumor of the lower ulna developed in the portion of the bone away from the former tumor of the radius. The lower segment of the ulna was removed and the histopathologic report indicated "osteogenic sarcoma of bone or giant cell tumor undergoing malignant change."

Jaffe, Lichtenstein and Portis[15] in 1940 reported 3 cases of malignant degeneration in a series of 14 cases of giant cell tumors of bone. One patient died with pulmonary metastases two years after curetting, there being no history of irradiation. The second patient had originally curettement and roentgen therapy, but five years later he, too, died with pulmonary metastases following amputation of the affected lower limb, which had shown a large recrudescence of the growth. In the third patient death resulted six years later, the treatment consisting of roentgen therapy alone. On the basis of these observations, Jaffe, Lichtenstein and Portis state that "there has been an apparent increase in the number of aggressive and malignant metastasizing giant cell tumors since the era of radiation therapy began." It is "not intended to imply that carefully controlled therapeutic irradiation of a giant cell tumor is neces-

sarily hazardous in all instances, but it is disturbing enough to suggest that the problem requires renewed considerations." Jaffe[16] later reported another case of bone cyst which six years after radiation therapy developed fibrosarcoma in the irradiated lesion.

Gershon-Cohen[11] in 1943 published the late results in a series of 29 cases of giant cell tumor of bone. Of these 14 were treated by radiation therapy alone and 14 by a combination of surgery and radiation therapy. Malignant degeneration developed in 2 cases treated by radiation therapy alone, in one six years and the other nine years after treatment. It was estimated that a total dose of 1,200 r and 6,400 r in air was given respectively.

Andersen[1] in 1945 reviewed 47 cases of giant cell tumor and osteitis fibrosa, observed at the Radium Center and the Surgical Service of the Finsen Institute in Copenhagen. A proved malignant degeneration was observed in only 1 case which was treated with 6,500 r, the roentgen irradiation constituting the sole method of treatment. In another case an osteogenic sarcoma was probably present from the beginning but remained undetected. This case will be discussed in greater detail later.

Haggart and Hare[13] treated 7 cases of large benign giant cell tumors of bone by a combination of preoperative roentgen therapy and thorough removal of the tumor eight or twelve weeks later. The remaining cavity was packed with bone grafts. A good result was obtained in every case, the average time since treatment being 4.7 years and the longest nine years and eight months. No malignant degeneration was noted.

Very recently Cahan, Woodard, Higinbotham, Stewart and Coley[3] reported 11 cases in which osteogenic sarcoma developed in irradiated bones six to twenty-four years after roentgen or gamma-ray therapy. Of these, 9 represented malignant degenerations of formerly benign giant cell tumors. In 5 cases there was microscopic proof of the benign character of the original bone lesion, whereas in 4 cases a benign character was assumed on the basis of roentgenographic study. In all instances radiation therapy constituted the most important phase of the treatment. The remaining 2 cases were examples of bone sarcoma developing in bone underlying areas irradiated for some soft tissue lesion. One case was that of a mammary carcinoma which was irradiated following radical mastectomy. Nearly eight years later she had a malignant melanoma of the skin overlying the right deltoid muscle which was removed, together with the axillary lymph nodes. After another three years a mass in the left upper anterior chest developed. A biopsy showed that this new mass was an "osteogenic sarcoma following roentgen irradiation of bone and not a metastasis from either the breast carcinoma or the melanoma." The other case was a little girl who originally had a retinoblastoma of the right eye, which was enucleated, and six months later a retinoblastoma of the left eye which was treated by intensive roentgen therapy. Five years after the irradiation she developed an osteogenic sarcoma of the frontal plate of the left orbit. This extended to both orbits, the nasal cavity and the cranial cavity, from which the patient very soon died. The right orbital plate remained free of tumor. The authors, on the basis of these observations, concluded that "roentgen therapy is not ordinarily to be recommended for nonmalignant tumors of bone."

In our own series there are 4 cases in which a malignant change was found in bone lesions originally diagnosed as benign, following roentgen therapy with ordinary technique. In Case II, however, the sarcomatous neoplasia was observed during the actual course of the treatments, so that the lesion was probably malignant from the beginning, and in Case III the roentgen dose was so small that its significance can almost certainly be disregarded. In the remaining 2 cases repeated pathologic fractures complicated the picture.

CASE II. E. F., white female, was first seen at

Harper Hospital on June 28, 1944, at the age of thirty-one years.

In March, 1944, the patient fell while working at her machine and sprained her right wrist. The injury was repeated five weeks later. She was examined in several clinics and a diagnosis of benign giant cell tumor of the lower end of the radius was made.

When seen at Harper Hospital there was a fusiform swelling of the dorsal aspect of the lower third of the right forearm, without discoloration of the skin or tenderness. Roentgenograms showed a cystic tumor of the lower third of the ulnar half of the radius with expansion of the bony cortex, suggestive of a benign giant cell tumor of bone (Fig. 3*A*).

On July 13, 1944, an operation was performed. The tumor area was incised posteriorly and thoroughly curetted. The cortex of the ulnar aspect of the radius was found to be of only paper shell thickness, but the articular plate was firm. The findings at operation seemed to confirm the clinical diagnosis of benign giant cell tumor.

The histopathologic report likewise was that of a "typical giant cell tumor with areas of new bone formation" (Fig. 4*A*).

From July 24, 1944, to March 1, 1945, four series of supervoltage roentgen treatments were given with a technique as practiced in benign giant cell tumors. The factors were: 500 kv. (constant), 7 mm. Cu, 3 mm. Al and 3 mm. celluloid as filters; half-value layer of 9 mm. Cu and an intensity of 20 r per minute. The dose for each series amounted to 70 per cent skin unit dose, two opposite portals being used for cross-firing.

After the last series of irradiation a recurrence of the swelling of the lower forearm was noted. A roentgen study made on May 11, 1945, showed a very definite expansion and extension of the lesion of the lower end of the radius (Fig. 3*B*).

On May 18, 1945, a second operation was performed. The radius was disarticulated at the elbow and the ulna was amputated in its middle third.

The microscopic examination of the specimen revealed an "osteolytic spindle cell sarcoma arising in a giant cell tumor of bone" (Fig. 4*B*).

From May 22 to May 26, 1945 the patient received a full course of prophylactic deep roentgen therapy over the lungs.

The last examination made on July 30, 1948, showed no metastases from the former bone sarcoma.

FIG. 3. Case II. *A*, giant cell tumor of the lower third of the radius; *B*, almost one year later, showing malignant change.

CASE III. J. D., white male, three years old when first referred to Harper Hospital on June 3, 1943.

The mother stated that in April, 1942, the patient fell and fractured the left femur. He was taken to another hospital where a roentgen examination revealed a spiral oblique fracture through a cystic area in the shaft of the left femur (Fig. 5*A*). Preliminary roentgenograms of other bones were negative. The left lower extremity was placed in a Bryant's traction for six days, then in a cast of plaster of paris for four months. After removal of this cast the child resumed his normal activity.

In December, 1942, the patient again fell and

a re-fracture through the same site resulted (Fig. 5*B*). At this time an open reduction and curettement was done, following which the extremity was placed in a Bryant's traction for six weeks and in a cast of plaster of paris for another six weeks.

The microscopic examination of the curetted material showed "young fibrous connective tissue with spicules of bone and cartilage; in a few areas giant cells of the epulis type were found. Final diagnosis: Bone cyst" (Fig. 7*A*).

In March, 1943, the patient was allowed to be up and about. The thigh externally appeared normal but the child had a peculiar limp when walking.

On June 8, 1943, roentgen study revealed rather dense healing of the fracture through the pathologic area of the mid-shaft of the left femur with good alignment. However, a cyst measuring 1.5 by 2 cm. in diameter was still present producing a slight expansion of the cortex (Fig. 6*A*). For this reason deep roentgen therapy was recommended.

The patient received two small roentgen exposures at intervals of two months with the following factors: 200 kv., 1.5 mm. Cu and 1 mm. Al filters; half-value layer of 1.9 mm. Cu and an intensity of 12 r/min. On June 9, 1943, a dose of 120 r in air was given over the left mid-thigh anteriorly and on August 11, 1943, a dose of 144 r in air posteriorly.

A roentgen examination made on November 11, 1943, showed excellent healing with complete reossification (Fig. 6*B*).

The patient continued to enjoy good health until the beginning of 1948 when he started to have pain at the site of the original lesion. This was followed by a progressive swelling of the left mid-thigh and difficulty in walking.

Re-examination made on March 3, 1948, revealed a definite tumor the size of a fist located mostly at the inner aspect of the left mid-thigh and intimately associated with the bone. Roentgen study showed a typical osteogenic sarcoma involving the mid-shaft of the left femur at the site of the former bone cyst (Fig. 6*C*).

On March 12, 1948, the malignant nature of the tumor was confirmed by biopsy and an immediate disarticulation at the hip joint was done.

The histopathologic report of the removed

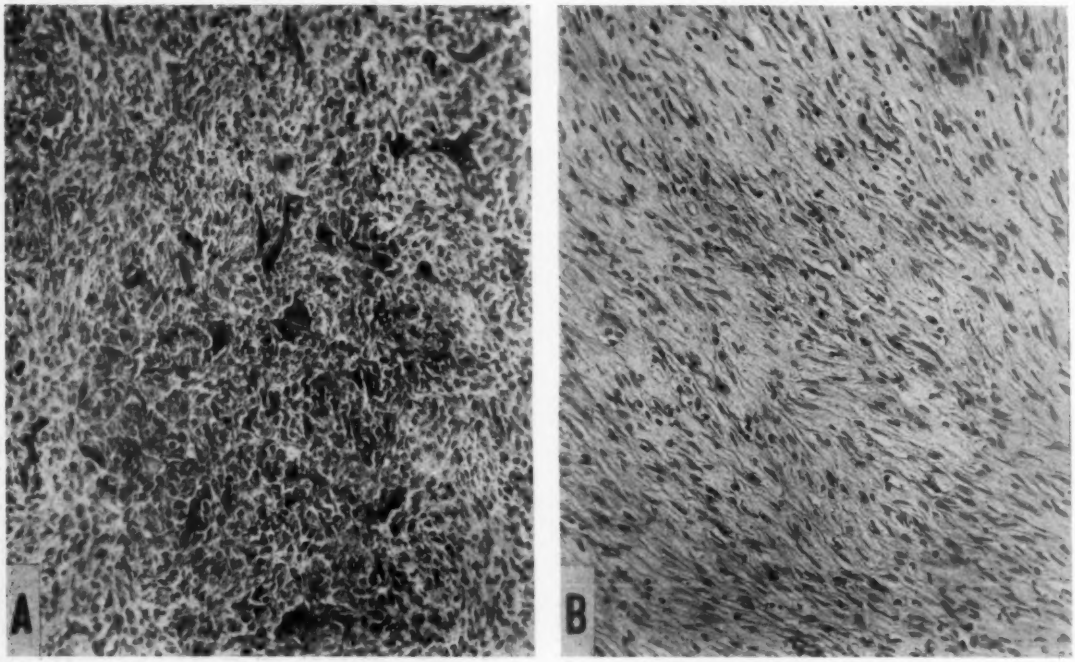

FIG. 4. Case 11. *A*, benign giant cell tumor; *B*, osteolytic spindle cell sarcoma arising in a giant cell tumor.

specimen was that of osteogenic sarcoma (Fig. 7*B*).

From March 29 to April 2, 1948, the patient received a course of prophylactic deep roentgen therapy over the lungs.

The last examination made in August, 1948, revealed no further manifestations of the osteogenic sarcoma.

CASE IV. W. C., white male, was admitted to Harper Hospital on March 12, 1936, at the age of thirty-five years.

In December, 1934, the patient sustained an intercondylar fracture of the right femur. He was hospitalized at another institution for three months during which time the extremity was immobilized. The fracture healed and the patient returned to work.

On January 3, 1936, he was struck with a crowbar on the right knee and another fracture resulted. He was treated elsewhere for a while then transferred to Harper Hospital for radiation therapy.

The examination made at the time of the admission revealed a swelling just above the right knee, the circumference of the limb being 4 cm. greater than that of the opposite side. A roentgen examination showed a multilocular, cystic tumor of the lower end of the right femur with a pathologic fracture through the involved area (Fig. 8*A*). The former roentgenograms made since December, 1934, were compared with the present ones. It was noted that a cystic area was already present at the original examination, it having been diagnosed as a "bone cyst." In view of the subsequent developments and the later roentgen appearance, the diagnosis was changed to that of benign giant cell tumor of bone. No biopsy was taken.

From March 13, 1936, to November 15, 1937, the patient received ten series of deep roentgen therapy with a technique such as practiced in giant cell tumor of bone. The factors were: 200 kv., 1.5 mm. Cu and 1 mm. Al filters, half-value layer of 1.9 mm. Cu and an intensity of 12–15 r/min. The initial dose amounted to 70 per cent skin unit dose, two portals being used for cross-firing. This was gradually reduced for each of the following series, until at the tenth series 10 per cent SUD was given.

The fracture continued to heal satisfactorily but on June 20, 1936, the patient re-freactured it a third time, necessitating hospitalization for three months.

FIG. 5. Case III. *A*, spiral fracture through a cyst in the shaft of the left femur; *B*, eight months later, showing refracture.

Finally, by the end of 1937, there was an excellent healing of the lesion and the patient was walking without difficulty.

A roentgen re-examination made May 20, 1941, showed a satisfactory result (Fig. 8*B*).

On March 28, 1942, the patient, while on a fishing trip, fell and at this time fractured his right patella which during the course of the former treatments became fixed to the callus formed during the reossification of the giant cell tumor. The patient was re-admitted to Harper Hospital. On April 27, 1942, the lower end of the patella was removed and a suprapatellar lysis was done.

Again the patient was able to resume his

FIG. 6. Case III. *A*, same as in Figure 5, before roentgen therapy (14 months after original lesion); *B*, five months after roentgen therapy; *C*, six years after original lesion, showing osteogenic sarcoma.

occupation and he continued to work regularly.

In July, 1944, an occasional vague pain occurred in the right knee. By February, 1945, this pain became quite steady at night and rather severe.

A checkup roentgen examination made on March 6, 1945, revealed a dissolution of the bone along the outer inferior aspect of the formerly involved area of the lower right femur, characteristic of malignant degeneration (Fig. 8*C*).

On March 19, 1945, a high amputation of the right lower extremity was performed.

The histopathologic examination of the specimen made by Dr. Leo W. Walker showed osteogenic sarcoma (Fig. 9).

The patient died on July 19, 1945, no detailed data being available.

Case v. Ch. W., colored male, was first seen at Harper Hospital on February 17, 1937, at the age of thirty-one years.

The patient stated that on December 3, 1936, while working in a gas station, he fell and injured his right hip. He was admitted to another hospital where he stayed for two weeks. A diagnosis of benign giant cell tumor of the upper end of the shaft and the lower end of the neck of the right femur with pathologic fracture was made. The patient received one series of deep roentgen therapy. This was repeated six weeks later.

When examined at Harper Hospital, on February 17, 1937, the roentgenograms showed a large multilocular cystic tumor of the upper end of the shaft and lower end of the neck of the right femur with a fracture undergoing partial healing (Fig. 10*A*).

From February 17 to June 5, 1937, the patient received three series of supervoltage roentgen therapy. The factors were: 500 kv. (constant), 7 mm. Cu, 3 mm. Al and 3 mm. celluloid filters; half-value layer of 9 mm. Cu and an intensity of 20 r/min. A tumor dose of 70 per cent SUD was given on each occasion through three portals.

On June 18, 1937, the patient fell in a theater and sustained a second pathologic fracture. He was admitted to another hospital where traction was applied for one month. After that the limb was immobilized by a body cast and the patient discharged to his home.

A checkup roentgenogram on January 25, 1938, showed healing of the fracture with some angulation (Fig. 10*B*).

At this time the patient dropped out of sight and was not heard of until April 12, 1945, when he returned with a marked swelling of the right hip associated with extreme difficulty in walking. A roentgen study showed what appeared to be an advanced osteogenic sarcoma at the site of the former benign giant cell tumor of the right upper femur (Fig. 10*C*). A disarticulation of the right hip was recommended but the patient refused.

From April 17 to October 29, 1945, the patient received four additional series of supervoltage roentgen therapy with varying doses, merely for palliation.

A film taken June 25, 1946, showed slow increase in size of the tumor (Fig. 10*D*). At the present time the patient is still able to be up and around aided by a specially constructed brace.

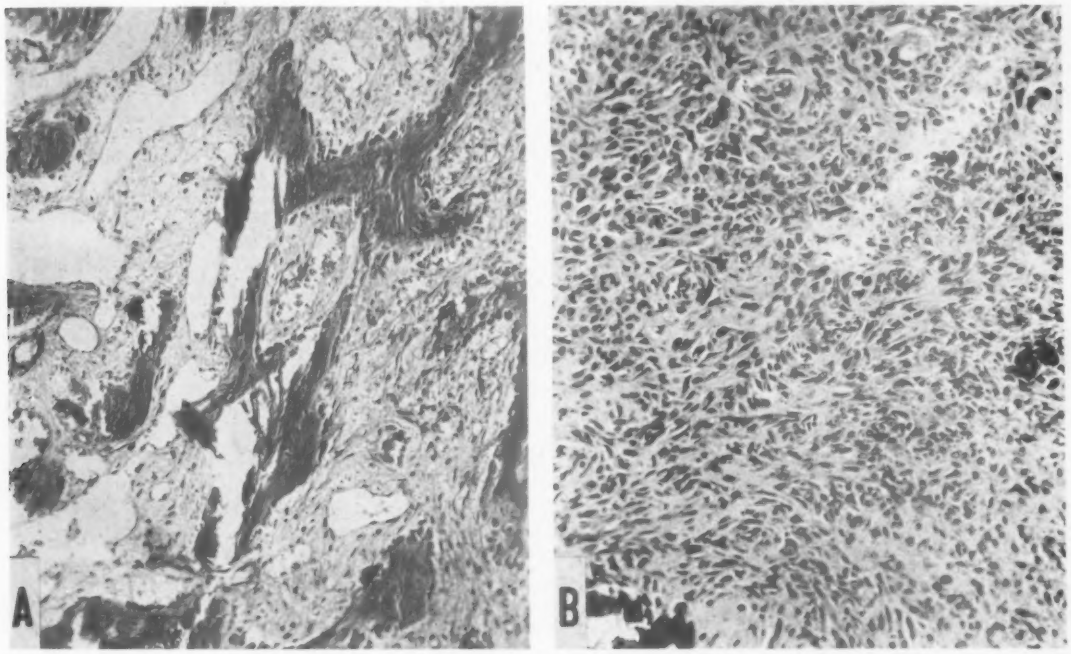

Fig. 7. Case III. *A*, bone cyst, photomicrograph of lesion shown in Figure 5*B*; *B*, osteogenic sarcoma, photomicrograph of lesion shown in Figure 6*C*.

FIG. 8. Case IV. *A*, benign giant cell tumor of the lower and of the right femur, with pathologic fracture; *B*, five years later showing good healing; *C*, nine years later showing malignant degeneration.

Spontaneous Malignant Degeneration of Benign Giant Cell Tumor. As a rule, the giant cell tumor, when allowed to progress to its natural termination, grows to large dimensions, necrosis and secondary infection complicating the picture in the later stages. Death results from hemorrhage or cachexia without evidence of regional or distant metastases.

For obvious reasons the literature is quite meager in cases showing a spontaneous malignant degeneration in benign giant cell tumors, without any treatment having been given. Kilgore and Abbott[17] in

1938 described a case in which a lesion in the center of the ulnar shaft was discovered while benign, remained benign for nearly six years, then turned into chondrosarcoma. An exploration two and one-half years after the first examination revealed a benign tumor "derived from cartilage rest of fetal type and accompanied by fibrous giant cell tissue and osteitis fibrosa." After the transformation into chondrosarcoma a resection of the affected portion of the ulnar shaft was performed. Four months later a destructive lesion of the radius at its midportion similar to the previous ulnar tumor was found. Immediately after this an amputation at the middle and lower thirds of the upper arm was done, but the patient died nine months later with distant metastases.

A very interesting case was reported in 1947 by Andersen.[1] A coal merchant, aged fifty-one, in October, 1935, presented himself for examination because of pain in the right knee of two years' duration. Roentgen study revealed a tumor in the outer condyle of the right femur which was interpreted as a benign giant cell tumor. No biopsy was taken. During a period of five months the patient was given 10,000 r to the right knee with apparently good effect. Three years later a recurrence developed and at this time a curettage was done. The histopathologic examination revealed osteitis fibrosa. Two years later a second recurrence appeared. Additional roentgenograms were made but no biopsy was taken. The condition still resembled the picture of benign giant cell tumor. Finally the next year—seven years after the onset of the disease—there was a typical osteogenic sarcoma. Further roentgen irradiation and amputation remained ineffective and the patient died six months later. The author in reviewing the case compared the microscopic slides from the curettage with those obtained from the amputation and arrived at the conclusion that the tumor was from the beginning "an only slightly malignant osteogenic sarcoma which for several years has masked itself as a benign giant cell tumor and in that time gradually has become highly malignant."

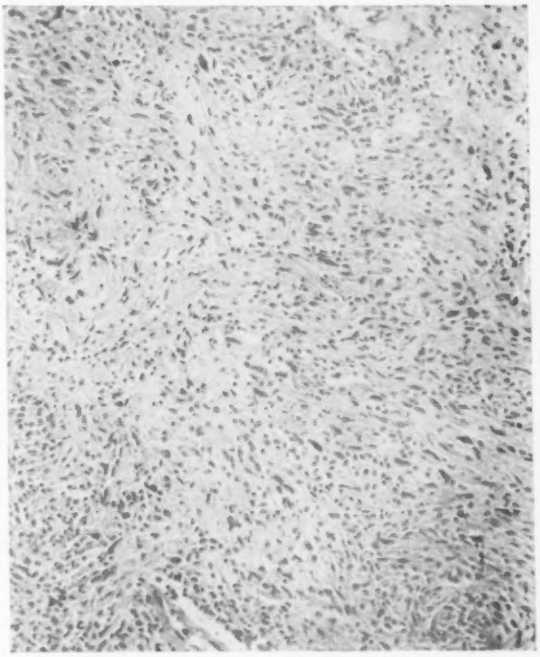

Fig. 9. Case iv. Osteogenic sarcoma; photomicrograph of lesion shown in Figure 8*C*.

There are a few other individual cases mentioned by various investigators in which apparently benign giant cell tumors after many years spontaneously changed into highly malignant sarcomas. Different theories have been advanced to explain this transformation. A number of investitors think, like Andersen, that such tumors are malignant to begin with but remain inactive for a shorter or longer period, then all at once exhibit a rapidly fatal malignant course. Others are of the opinion that the assumption of malignant characteristics actually represents metaplasia in a manner similar to that observed in osteochondromas, fibromas, myxomas and other benign tumors which are known not infrequently to become sarcomatous.

In our own series there is one case which might be interpreted as a spontaneous degeneration of a bone cyst into an osteogenic sarcoma.

Case vi. M. Sh., white female, aged sixty, was first admitted to Harper Hospital in May,

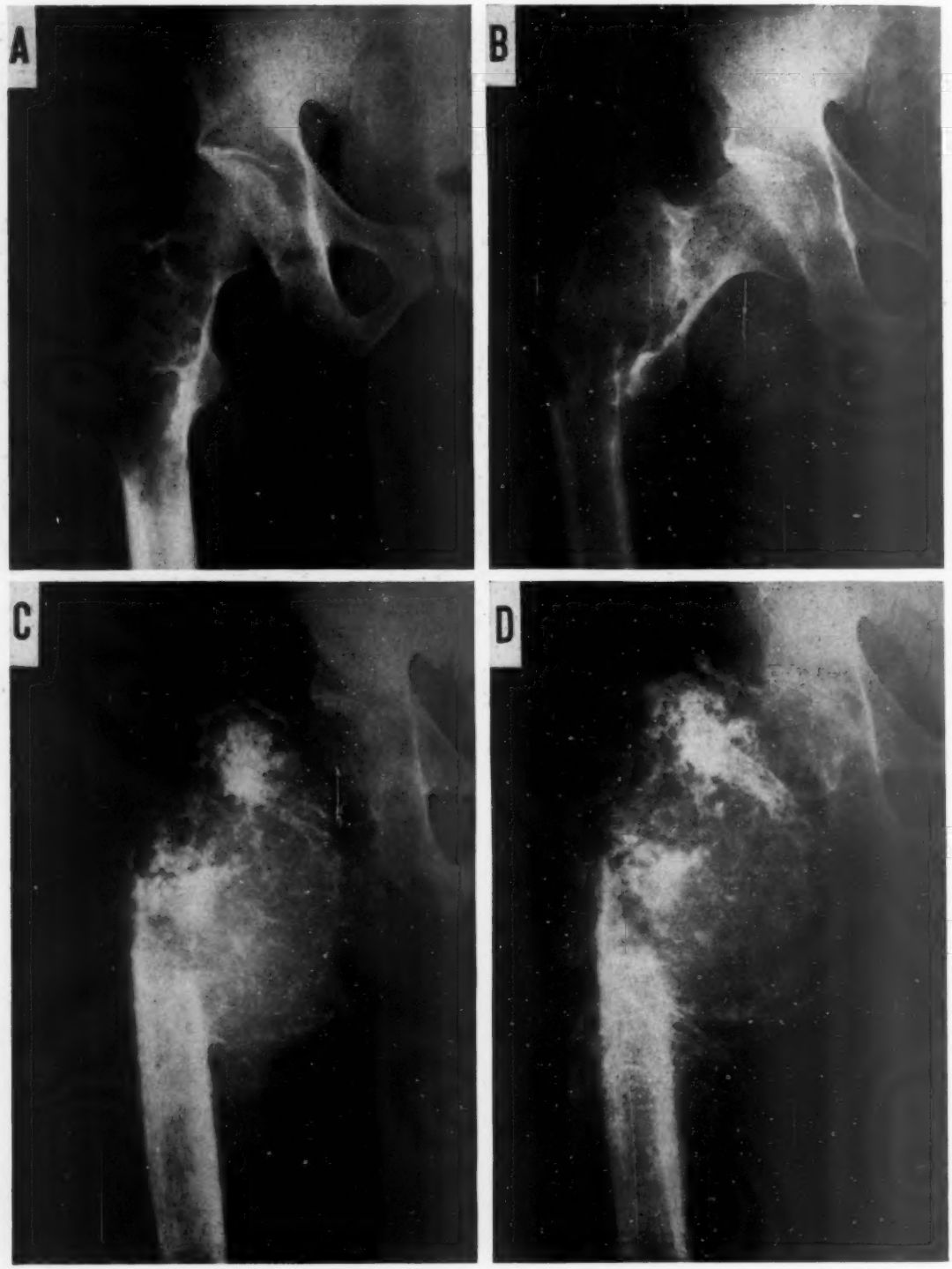

FIG. 10. Case v. *A*, benign giant cell tumor of the upper end of the left femur; *B*, one year later; *C*, eight years later showing malignant degeneration; *D*, nine years later.

1939, for treatment of extensive diverticulosis involving the sigmoid colon. At this time the patient also complained of pain in the right knee, with difficulty in walking, of several years' duration. A roentgen examination of the right knee made on May 19, 1939, revealed a

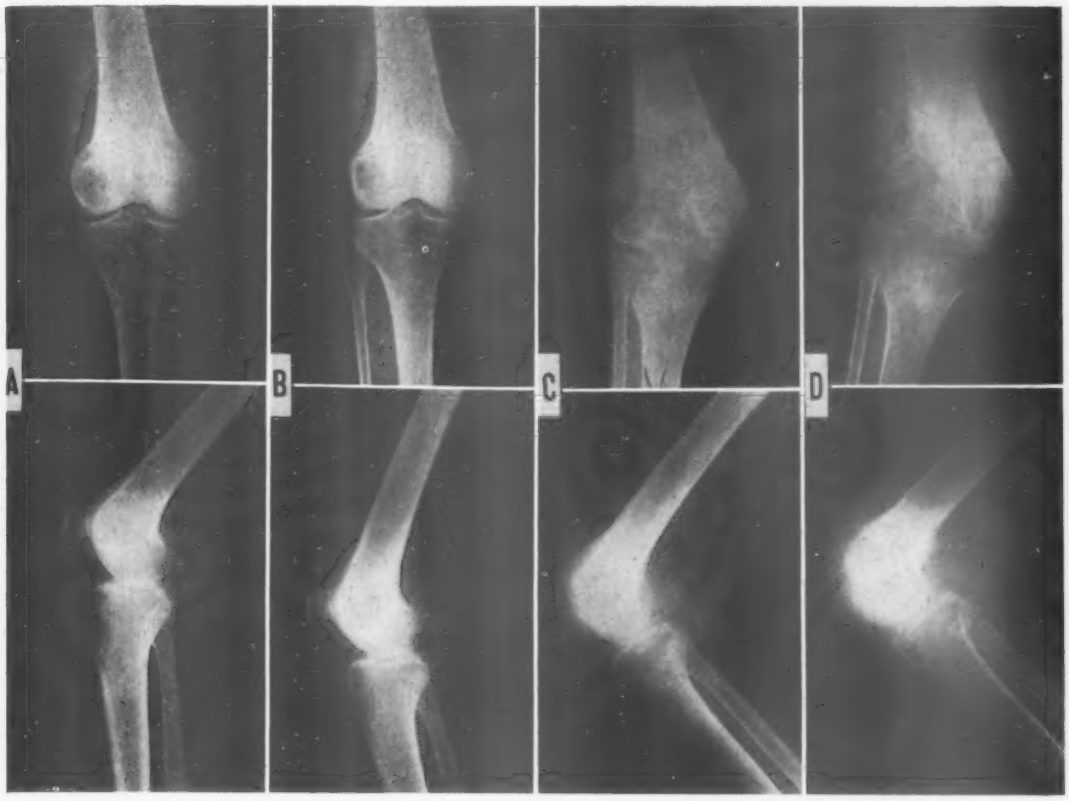

FIG. 11. Case VI. *A*, bone cyst of the external condyle of the right femur; *B*, one year later; *C*, three years later, interpreted as benign giant cell tumor; *D*, four and one-half years later showing malignant degeneration.

cystic area of the external condyle of the right femur (Fig. 11*A*). A biopsy was recommended but the patient refused.

After her discharge from Harper Hospital the patient consulted various physicians and had different types of physiotherapy, including electrical treatments and diathermy.

On May 22, 1940, she returned to Harper Hospital for a checkup examination. Roentgenograms revealed no change in the cystic area (Fig. 11*B*). Again a biopsy was recommended and refused.

The patient was then not seen until May 4, 1942, when she returned with a rather marked swelling of the right knee associated with very intense pain. She stated that for about one month she was forced to walk with the aid of crutches, being unable to put her weight on the right lower extremity. A re-examination at this time revealed a definite tumefaction just above the outer aspect of the right knee. Roentgenograms showed a lesion involving the entire external condyle and part of the lower shaft of the femur with an intercondyloid pathologic fracture (Fig. 11*C*). A clinical diagnosis of benign giant cell tumor of bone was made and radiation therapy recommended.

From May 19, 1942, to July 7, 1943, the patient received seven series of supervoltage roentgen therapy. The factors were: 500 kv. (constant), 7 mm. Cu, 3 mm. Al and 3 mm. celluloid filters; half-value layer of 9 mm. Cu, and an intensity of 20 r/min. The dose amounted to 70 per cent SUD, given through two opposite portals, for each of the series. First there was definite improvement but from the fifth series on it became increasingly evident that the condition represented a sarcoma (Fig. 11*D*). Surgical intervention was again recommended but the patient persistently refused.

Finally in November, 1943, she gave her consent to the operation and on November 10, 1943, an amputation at the junction of the upper and middle thirds of the right thigh was performed.

The histopathologic examination of the specimen revealed "osteogenic sarcoma" (Fig. 12).

Soon after the operation this patient moved

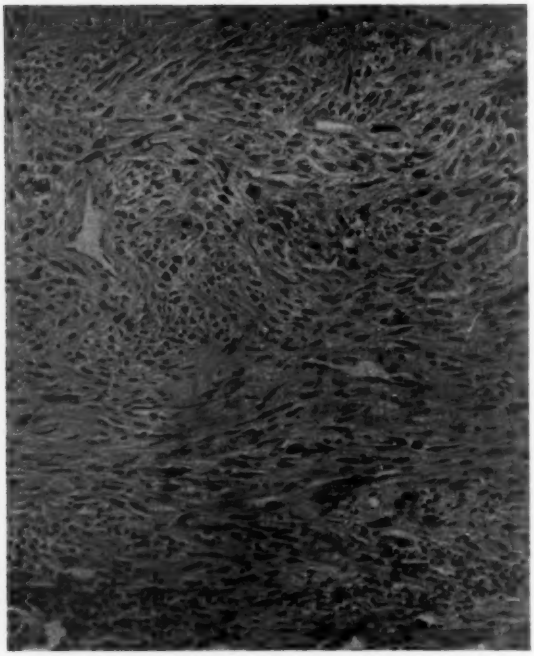

FIG. 12. Case VI. Osteogenic sarcoma; photomicrograph of lesion shown in Figure 11*D*.

and the final outcome of the case is therefore unknown.

Malignant Degeneration of Benign Giant Cell Tumor Following Surgery. There are a number of articles in the literature dealing with the subsequent occurrence of sarcoma or other malignant manifestation in cases of giant cell tumors of bone which were treated by surgery. The first of such articles is apparently that of Stone and Ewing[29] which was published in 1923 and which for many years has created considerable discussion. These authors refer to a young man who at the age of nineteen years became afflicted with a giant cell tumor of the head of the tibia. The lesion was treated by repeated curettage, one intracavitary application of radon seeds and later amputation. The patient died six months after the amputation with lung metastases. The next year MacGuire and McWhorter[19] reported a series of 20 cases of giant cell tumor, 4 of whom died with metastases to the lungs. Three were treated by curettage or amputation and in the fourth a combination of curettage, intracavitary radium and external roentgen therapy was used.

In 1931, Simmons[27] in a more comprehensive article reviewed 116 cases of benign giant cell tumor collected by the Registry of Bone Sarcoma of the American College of Surgeons. The cases were treated by various means prior to 1925 and 82 of them were followed up for a period of five years or longer. In the entire group 6 patients died from metastases. In the same year a single case who died with metastasis was also reported by Dyke.[8] The giant cell tumor was described as having resulted from injury. Following curettage and later amputation the patient developed metastases to the lungs and died.

W. B. Coley[6] in 1935 expressed the view that 15 per cent of those diagnosed as benign giant cell tumors by experienced pathologists later become malignant. He, personally, reported 19 cases. In 7 the treatment consisted of a combination of curettage and roentgen therapy, in another 4 of a combination of irradiation and late amputation, in 7 of surgery alone, and in 1 of roentgen therapy alone. In a later article, B. L. Coley and Higinbotham,[5] in reviewing their experience in 124 cases of giant cell tumor of the bone, state that "from a practical standpoint, one should bear in mind that infrequently, but by no means rarely, a giant cell tumor may exhibit all the characteristics of a benign lesion, even to the histological appearance, and yet run a course that shows it to be a malignant sarcoma." In this series 1 case came to amputation for local malignant change following surgery alone and 6 cases following surgery and radiation therapy combined.

A very instructive article was published in 1937 by Codman[4] who studied the result of various types of treatment in a group of 153 cases of giant cell tumor about the knee listed in the Registry of Bone Sarcoma of the American College of Surgeons from 1920 to 1936. Of these, 100 cases were followed up for five years or longer and 50 for ten years or longer. An additional group of 12 cases which were chosen from over 600 cases of malignant tumors of bone about the knee joint showed a great resemblance to giant cell tumor of bone

roentgenographically but biopsy proved them to be malignant. In the group of 153 cases which were classified as benign originally, 14 cases showed malignant characteristics later. According to Codman, "it would be unfair to say that they all became malignant for in most of the 14 cases the original slide was not available." Therefore, to gain a better idea about the possibility of malignant transformation, they were divided into two groups: (1) cases in which both roentgenograms and slides were interpreted as giant cell tumor yet the disease assumed a rapidly fatal course, all patients dying within five years; and (2) cases in which roentgenograms, slides and the clinical course for several years made the diagnosis seem assured, yet eventually sarcoma appeared at the site of lesion. There were 8 cases in the first group: in 5 the type of treatment was not specified and in the remaining 3 surgery alone was used (curettage followed by amputation in 1 and amputation in 2). The second group contained 6 cases: in 2 the treatment was not specified, in 1 surgery alone and in 3 roentgen therapy alone was used. The impression was gained by Codman that whereas in the first group an error of interpretation of the type of the original tumor may have taken place, in the latter group a malignant transformation of the benign giant cell tumor may be assumed.

Stewart, Coley and Farrow,[28] in 1938, reported 6 cases of giant cell tumor of bone which became malignant. Of these, 3 were treated by roentgen therapy alone, 2 by curettage, and the sixth represents the case of Stone and Ewing[29] already referred to. A seventh case was added which was found to be malignant from the beginning. In all cases, except the sixth, the malignant characteristics were discovered within one to three years after the original diagnosis. Therefore the impression was gained that "there are tumors which show malignant features from some early period but which are, to all intents and purposes, identical in nature with the benign giant cell tumor and which are distinct from osteogenic sarcoma in the accepted sense."

Meyerding[22] in 1941 reviewed 124 cases of giant cell tumors of bone which were observed at the Mayo Clinic from 1916 to 1940. He divided them into two groups: the benign giant cell tumor and the malignant giant cell sarcoma. Of the entire series 101 cases were classified as belonging to the first group with a five year cure of 97 per cent, and 23 as belonging to the second group with a five year cure of 65 per cent. The treatment consisted of various methods, among which surgery and radiation therapy, either alone or combined, occupied the most important part. On the basis of the study, Meyerding arrived at the conclusion that "in giant cell tumors that are apparently benign malignant changes may develop in time after surgical procedures or irradiation, or after a long period."

In 1945, Meyerding[23] published the results in 40 cases of benign giant cell tumor of bone treated from 1913 to 1943 by resection or excision and bone grafting. Of these, 26 patients had no previous treatment, 4 received only radiation therapy, 3 had some form of operation, 3 a combination of operation and radiation therapy, 2 excision and bone grafting and in 2 a diagnosis of sarcoma had been made and amputation advised. Three of the 40 patients died from incidental disease, 1 seven years after the operation. In the remaining, not only a cure but a good functional result was obtained. The conclusion was therefore reached that massive resection and excision by curettage followed by cauterization and filling of the resultant cavity with autogenous bone constitutes a safe procedure in properly selected cases.

A more radical surgical approach is recommended also by Mandl and Dwek.[20] These authors observed 2 cases of giant cell tumor of bone in which curettage was followed by the appearance of osteogenic sarcoma without the administration of roentgen therapy. In 1 case curettage was performed for a condition which roentgenographically was diagnosed as osteitis fibrosa of the tibia. The microscopic study indicated the presence of a giant cell tumor.

Three years later an osteogenic sarcoma developed and an amputation had to be done. In the second case a curettage for a giant cell tumor of the tibia was followed within a few months by a recurrence which proved to be osteogenic sarcoma. Because of this possible incompleteness of the curettage, Mandl and Dwek[20] recommended a more thorough excision or resection with insertion of a sliding bone graft.

Our series contains 2 cases in which the sarcomatous change developed following surgical procedures. In one case three surgical interventions were followed by roentgen therapy and an osteogenic sarcoma was noted seven years later. In the other case the malignant degeneration followed curettement with grafting of bone chips. This latter patient was subsequently treated with roentgen therapy and a good result was obtained persisting now for more than three years.

CASE VII. S. L., white male, was first seen at Harper Hosptial on June 27, 1925, at the age of twenty-four years.

In the late fall of 1924 the patient noticed a swelling in the region of the upper third of the tibia.

A roentgen examination made on June 27, 1925 revealed a benign giant cell tumor of the right upper tibia (Fig. 13*A*).

From June, 1925, to September, 1927, the patient had two curettements at other institutions.

On September 10, 1927, a third curettement was done at Harper Hospital and the attending surgeon at this time used bismuth paste to fill the bone cavity.

The microscopic report was that of benign giant cell tumor of bone (Fig. 14*A*).

On September 26, 1927, the patient was referred for deep roentgen therapy because of the persistent recurrence of the lesion. A preliminary roentgenogram showed the bismuth paste completely filling the bone cavity (Fig. 13*B*).

From September 26, 1927, to April 5, 1928,

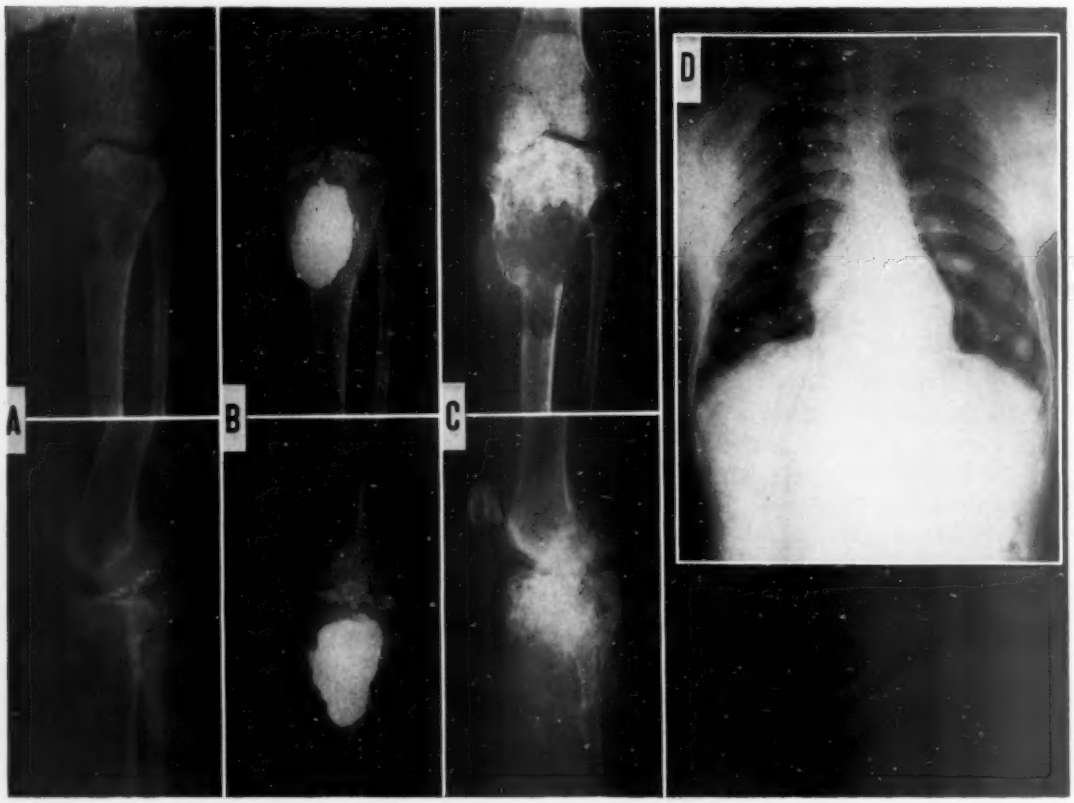

FIG. 13. Case VII. *A*, benign giant cell tumor of the upper third of the right tibia; *B*, two years later, bismuth paste filling the bone cavity; *C*, seven years later, showing malignant degeneration; *D*, metastases to lungs.

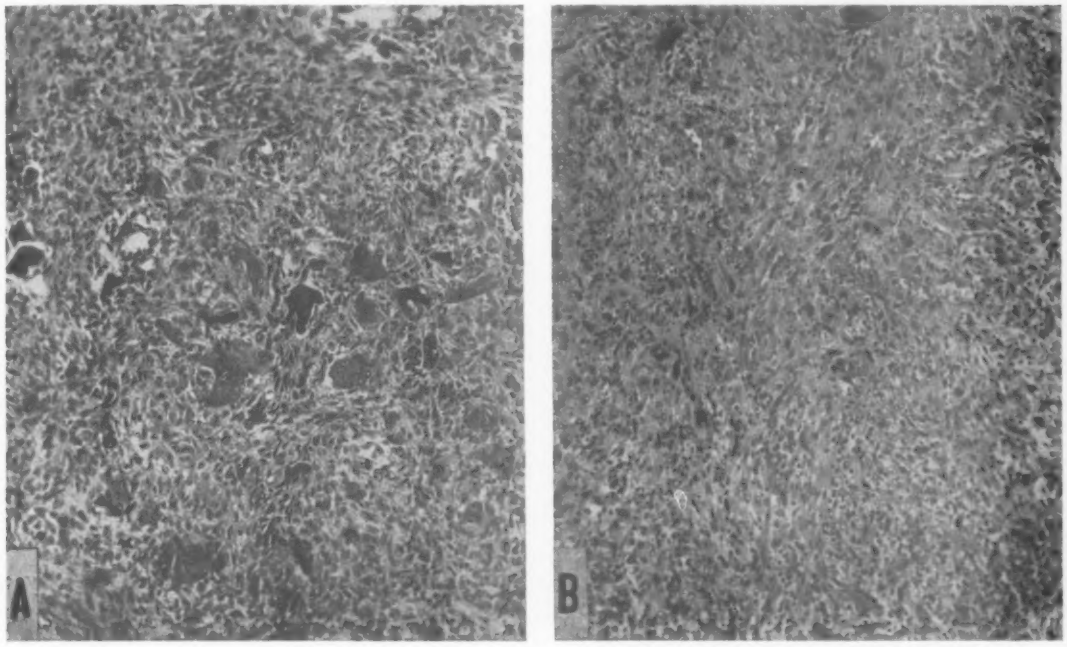

Fig. 14. Case vii. *A*, benign giant cell tumor of bone; photomicrograph of lesion shown in Figure 13*A*; *B*, osteogenic sarcoma; photomicrograph of lesion shown in Figure 13*C*.

the patient received five series of deep roentgen therapy. The factors were: 200 kv., 1 mm. Cu and 1 mm. Al filters; half-value layer of 1.8 mm. Cu and an intensity of 20 r/min. The initial tumor dose amounted to 90 per cent SUD, two opposite portals being used for cross-firing. The subsequent tumor doses were gradually reduced down to 50 per cent SUD at the fifth series.

The patient completely recovered and felt well until the spring of 1932 when he noticed a slight swelling at the site of the original tumor of the right tibia. On July 21, 1932, he fell and suffered a fracture through the upper end of the right tibia. A roentgen examination at this time revealed changes suggestive of a malignant degeneration of the former benign giant cell tumor complicated by a pathologic fracture (Fig. 13*C*).

On August 29, 1932, the tumor area was curetted for the fourth time. Simultaneously a parathyroidectomy was performed on the assumption that benign giant cell tumor of bone represents a solitary lesion of a Recklinghausen's disease incidental to hyperparathyroidism.

The microscopic examination of the tissue curetted from the tumor area showed "rapidly growing and markedly anaplastic polymorphous cell sarcoma" (Fig. 14*B*).

Soon after the operation metastases developed in the lungs (Fig. 13*D*) and the patient died in January, 1933,

Case viii. E. M. S., white female, aged thirty-seven, was first admitted to Harper Hospital on April 8, 1944,

The patient stated that on March 19, 1942, she tripped over a bag of grass seed and fell fracturing her left upper femur. She was taken to another hospital where she stayed for over three months. Traction was applied for six weeks and a body cast for another seven weeks. Then a brace was supplied and the patient was allowed to walk with the aid of crutches.

A roentgen examination at the time of admission to Harper Hospital revealed the presence of a multilocular cystic lesion in the upper end of the left femoral shaft, suggestive of benign giant cell tumor of bone (Fig. 15*A*).

On April 10, 1944, an operation was performed. An incision was made through the lateral aspect of the involved part of the upper femur and a window cut in the greatly thinned cortex of the bone. A moderate quantity of orange colored fluid was released exposing an irregular cavity about 3 inches long and 1½ inches in diameter, which was thought to be a simple bone cyst. The cavity was completely filled with bone chips removed from the left anterior iliac crest.

The microscopic examination indicated "bone cyst with giant cells" (Fig. 16*A*).

Subsequent roentgen study made on June 7, 1944, showed a very satisfactory operative result (Fig. 15*B*).

Additional checkup roentgenograms were taken at intervals of two to three months. The good result continued until May 31, 1945.

At this latter examination a definite expansion of the involved part of the femoral shaft was noted with a rather diffuse area of bone destruction suggestive of malignant metaplasia (Fig. 15*C*).

On July 16, 1945, a second operation was performed. The involved area of the left femoral shaft was explored and thoroughly curetted.

The microscopic report at this time was that of an osteolytic sarcoma of bone with a few giant cells (Fig. 16*B*).

Because of the above pathologic findings radiation therapy was recommended.

From July 30 to December 19, 1945, the patient received three series of supervoltage roentgen therapy with a technique as practiced in the osteolytic type of sarcoma of the bone. The factors were 500 kv. (constant), 7 mm. Cu, 3 mm. Al and 3 mm. celluloid as filters; half-value layer of 9 mm. Cu and an intensity of 20 r/min. Three portals of entry were used for cross-firing, one portal being treated per day with a full dose. The tumor dose for the three series amounted to 130 per cent, 110 per cent and 90 per cent SUD respectively.

Subsequent checkup roentgenograms revealed a satisfactory result. At the present time the patient is walking normally and there is a good reossification of the bone (Fig. 15*D*).

SUMMARY AND CONCLUSIONS

During a period of twenty-five years (1923 to 1947, inclusive) a series of seventy-

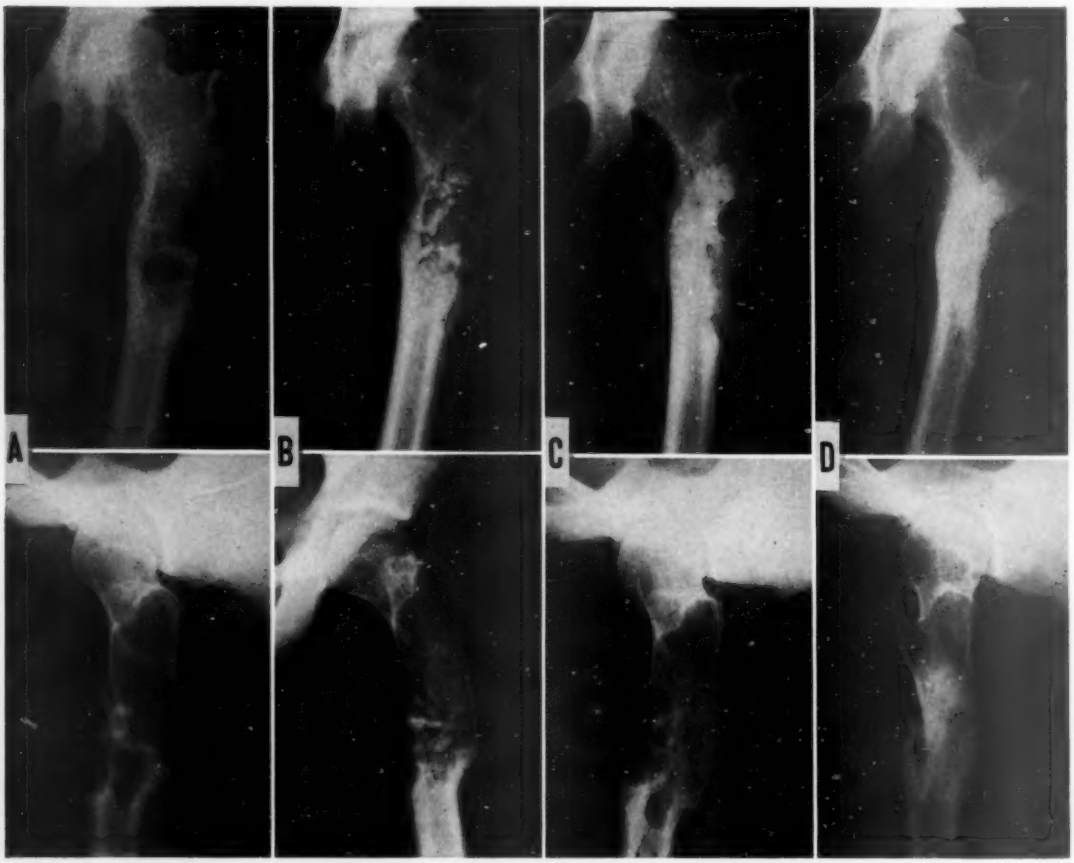

FIG. 15. Case VIII. *A*, multilocular cyst of the upper end of the left femur; *B*, after curettage and filling of cavity with bone chips; *C*, one year later, showing malignant metaplasia; *D*, five years later (three years after roentgen therapy) showing satisfactory result.

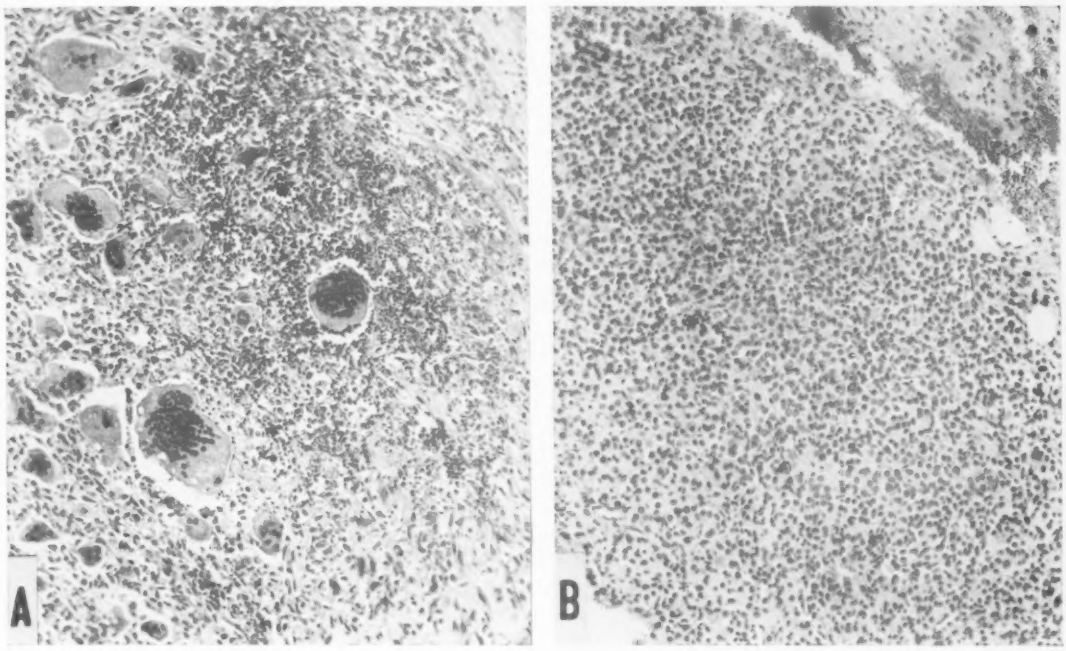

FIG. 16. Case VIII. *A*, bone cyst with giant cells; photomicrograph of lesion shown in Figure 15*A*; *B*, malignant metaplasia; photomicrograph of lesion shown in Figure 15*C*.

seven cases of benign giant cell tumor of bone were treated at Harper Hospital, Detroit, mostly by roentgen therapy. Five of the cases are known to be dead, three from incidental disease and two as a result of subsequent sarcomatous change of the benign giant cell tumor. Five cases are untraced, one of which had a malignant degeneration of the benign giant cell tumor. In an additional four cases a sarcomatous change of the benign giant cell tumor developed but after further treatment, either by surgery or irradiation, a satisfactory final result was obtained.

The seven cases in which the malignant degeneration occurred are presented in detail. They represented 9 per cent of the total of seventy-seven cases of the series.

In four cases the malignant degeneration was noted after routine roentgen therapy. However, in one the sarcomatous neoplasia was observed before the contemplated series of treatments were concluded so that the lesion was probably malignant from the beginning. In another, the roentgen dose given was so small that its significance can almost certainly be disregarded. In the remaining two cases repeated pathologic fractures complicated the picture.

In one case the malignant degeneration apparently was spontaneous.

In two cases the sarcomatous change developed following surgical procedures. In one of these three surgical interventions were followed by roentgen therapy and an osteogenic sarcoma was noted seven years later. In the other case the malignant metaplasia followed curettement with filling of the cavity with bone chips.

An eighth case is included in which fibrosarcoma of the soft tissues and sternum developed eighteen years after gross overirradiation for thymic enlargement.

Thus it may be said that in the enumerated cases the malignant degeneration resulted: (1) spontaneously; (2) following repeated surgery and irradiation; (3) following roentgen therapy alone; (4) following surgery alone when bone chips were used to fill the bone cavity.

The conclusion is drawn that the malignant degeneration of the giant cell tumor of bone occurs in a limited number of cases (10–15 per cent) as a natural sequence of

events unaffected by the type of treatment given.

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THE VALUE OF UPRIGHT ROENTGENOGRAMS OF THE MATERNAL PELVIS AT OR NEAR TERM WITH PARTICULAR REFERENCE TO PLACENTA PRAEVIA*

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THE purpose of this paper is to emphasize the value of upright roentgenograms of the maternal pelvis recommended by Ball and Golden.[1] They observed that in normal cephalic presentations the fetal head dips into the pelvic inlet and occupies the mid-coronal and mid-sagittal planes of the inlet during the last trimester of pregnancy when the mother is upright (Fig. 1). However, with the mother horizontal the fetal head shifts in relation to the pelvic inlet with change in position of the maternal pelvis. Any pelvic mass in addition to placenta praevia, either marginal or central, may displace the fetal head from the central position or prevent it from entering the pelvic inlet (Fig. 2, 3 and 4).

The relation of the size of the fetal head to the pelvic inlet is usually apparent by inspection of the films since both structures are in more or less the same plane.

For the past three years all pregnant patients at Garfield Memorial Hospital have been examined in the following manner:

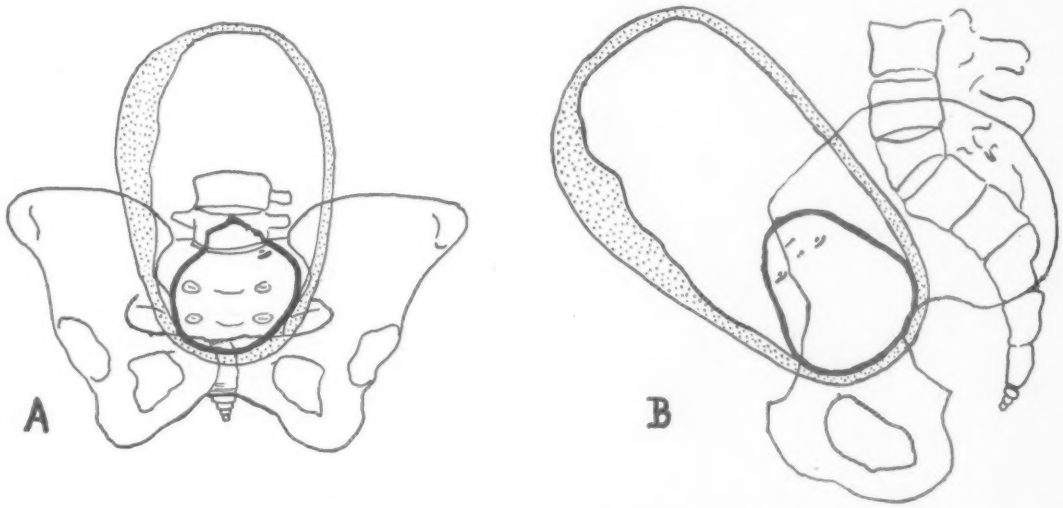

FIG. 1. Sketches of anteroposterior and lateral views of the maternal pelvis with patient upright. Normal findings. (*A*) The fetal head is centrally located. (*B*) The fetal head is centrally located and is engaged. The head is equidistant from the anterior margin of the first sacral segment and from the posterior margin of the symphysis.

1. Lateral and posteroanterior roentgenograms are made with the patient horizontal and with the central ray directed to the center of the pregnant uterus. These films are made at a target-film distance of 30 inches with a movable grid and the shortest possible exposure time. Pillows may be placed under the thighs and chest

[1] Ball, R. P., and Golden, R. Roentgenographic obstetrical pelvicephalometry in the erect posture. AM. J. ROENTGENOL. & RAD. THERAPY, 1943, *49*, 731–741.

* From the Radiological Clinic of Drs. Groover, Christie and Merritt, Garfield Memorial Hospital, Washington, D. C.

to make the patient more comfortable and instructions given for the patient to take several deep breaths before exposure to minimize motion of the patient and fetus.

roentgenograms also demonstrate fetal structure to great advantage. This examination may be made with the patient upright, if an upright movable grid is avail-

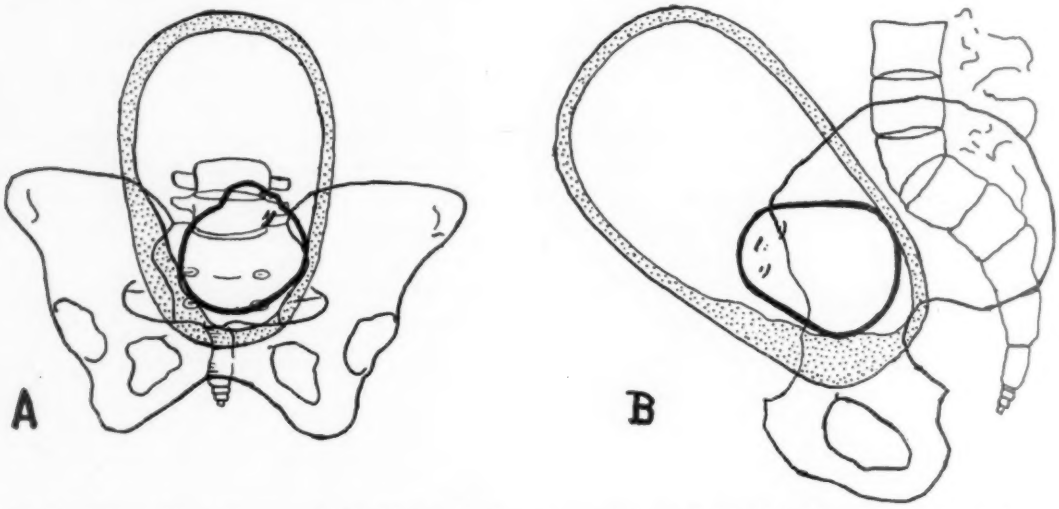

FIG. 2. Sketches with patient upright. Marginal placenta praevia. (*A*) The fetal head is displaced laterally. (*B*) The fetal head is displaced posteriorly by the placenta which is located on the anterior wall of the uterus. Contrast medium in the bladder is of value when the placenta is located anteriorly.

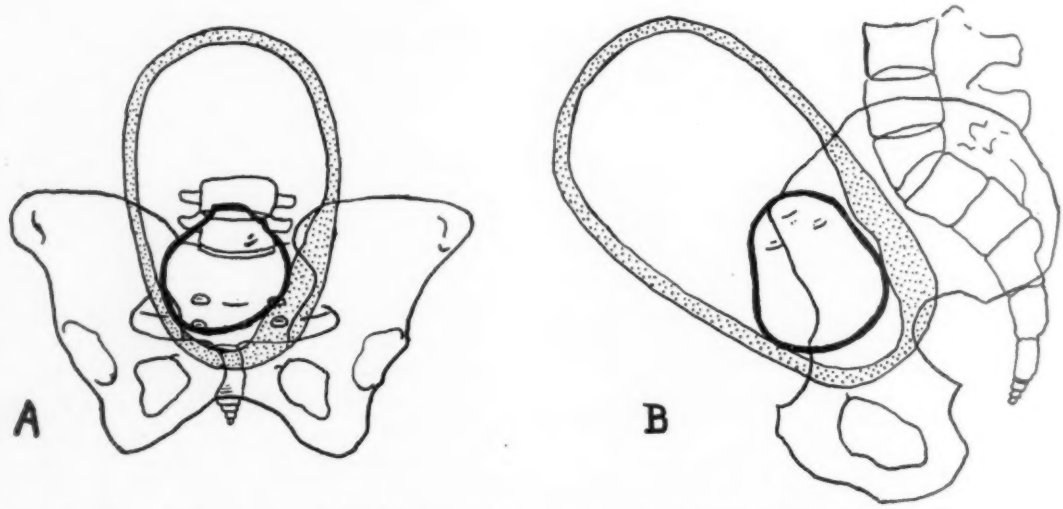

FIG. 3. Sketches with patient upright. Marginal placenta praevia. (*A*) The fetal head is displaced laterally. (*B*) The fetal head is displaced anteriorly by the placenta which is located on the posterior wall of the uterus. Contrast medium in the rectum is of value when the placenta is located posteriorly.

Such films provide adequate visualization of the upper three-fourths of the uterus and usually serve to visualize the placenta or a portion of it. Oblique views of the uterus may occasionally be necessary to definitely outline the placenta. These soft tissue

able, since it is more comfortable for the patient than the horizontal position.

2. Lateral and anteroposterior roentgenograms are made with the patient upright and with the central ray directed through the pelvis 1 inch above the level of

the greater trochanter of the femur. These films may be made at any distance with a movable or stationary grid and provide adequate visualization of the maternal pelvis and fetal head rather than soft tissue detail.

Thirty cases suspected of having placenta praevia have been examined in the Department of Radiology during the past three years. Twelve of these patients had abnormal findings indicative of placenta praevia and these findings were subsequently confirmed at time of operation. Two patients had findings indicative of cephalopelvic disproportion. From these 30 cases several observations have been made which verify those first reported by Ball and Golden. These observations may be grouped as follows:

1. If the placenta is visualized with certainty in the upper portion of the uterus and the head enters or centers upon the pelvic inlet with the patient upright, there is no evidence of placenta praevia or cephalopelvic disproportion (Fig. 5).

2. If the placenta is visualized in the mid or lower portion of the uterus and the head is displaced in relation to the inlet with the patient upright, the displacement is attributed to marginal location of the placenta (Fig. 6, 7, 8 and 9).

3. Non-visualization of the placenta and upward displacement of the head in the absence of cephalopelvic disproportion is attributed to a centrally located placenta.

4. Failure of the head to enter or center upon the inlet may be due to disproportion of the fetal head in relation to the inlet and should be apparent from the measurement of the size of the head and the inlet.

5. Visualization of the placenta in the upper portion of the uterus with displacement of the head in relation to the inlet and without evidence of disproportion is indicative of a pelvic tumor.

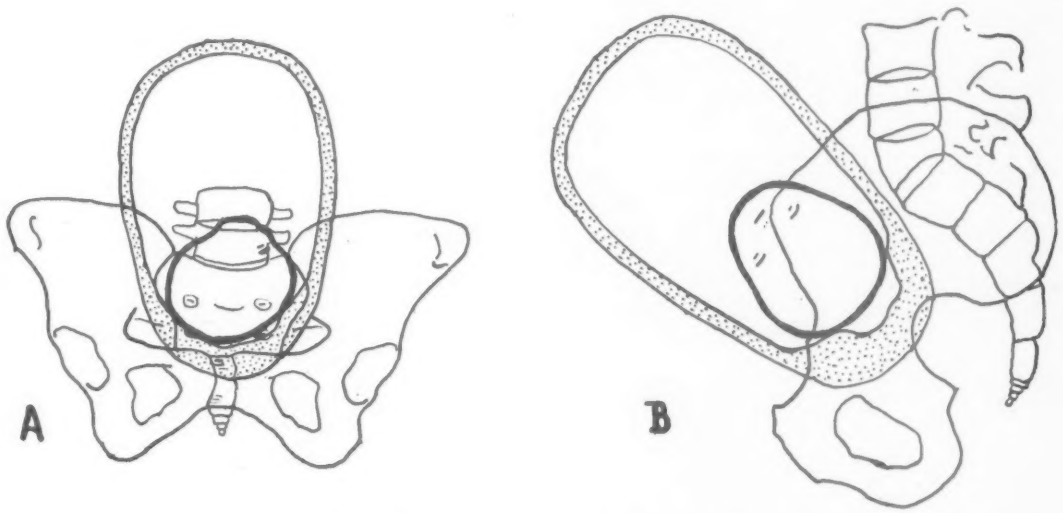

FIG. 4. Sketches with patient upright. Central placenta praevia. (*A*) The fetal head is centrally located but appears high. (*B*) The fetal head is centrally located but is not engaged. Contrast medium in the bladder and rectum of value when the placenta is centrally located.

In one patient the placenta was visualized in the upper portion of the uterus and yet the fetal head failed to engage with the patient upright in spite of more than adequate inlet measurements. A normal delivery followed. For this we have no explanation but in our experience its occurrence is rare.

6. Indefinite visualization of the placenta and upward displacement of the fetal head in the absence of cephalopelvic disproportion may be due to delayed rupture of the membranes. In such a case the fluid-

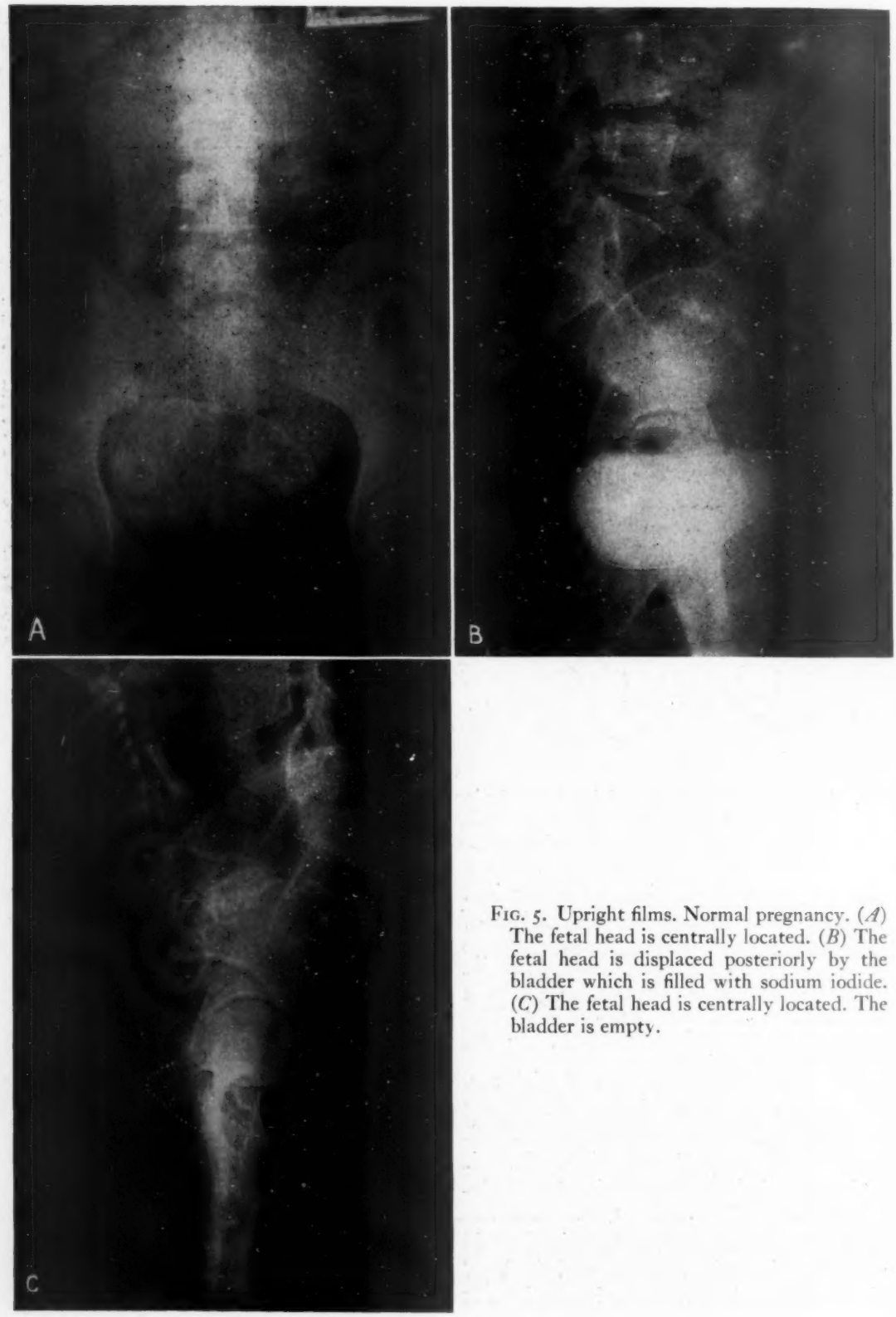

FIG. 5. Upright films. Normal pregnancy. (*A*) The fetal head is centrally located. (*B*) The fetal head is displaced posteriorly by the bladder which is filled with sodium iodide. (*C*) The fetal head is centrally located. The bladder is empty.

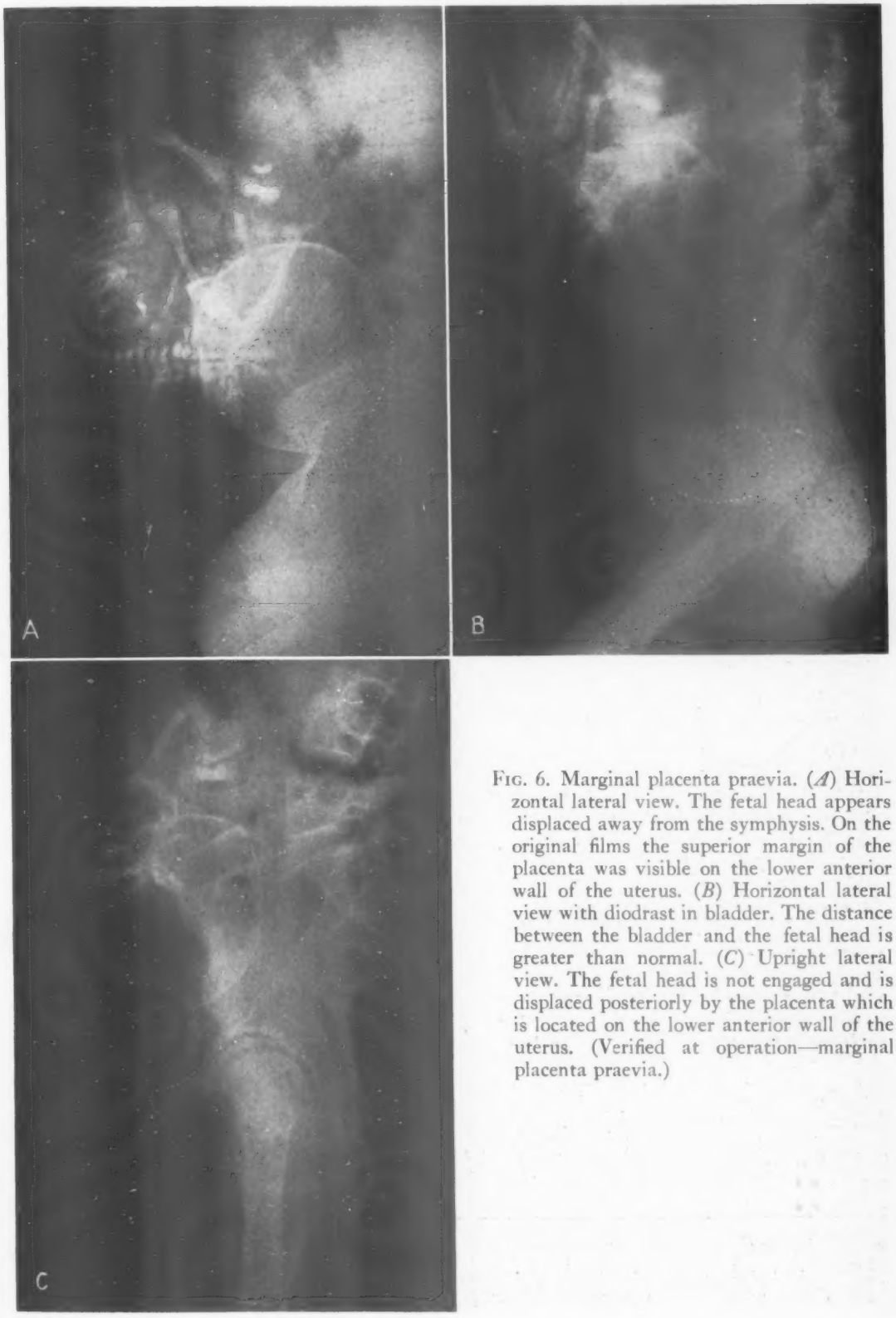

Fig. 6. Marginal placenta praevia. (*A*) Horizontal lateral view. The fetal head appears displaced away from the symphysis. On the original films the superior margin of the placenta was visible on the lower anterior wall of the uterus. (*B*) Horizontal lateral view with diodrast in bladder. The distance between the bladder and the fetal head is greater than normal. (*C*) Upright lateral view. The fetal head is not engaged and is displaced posteriorly by the placenta which is located on the lower anterior wall of the uterus. (Verified at operation—marginal placenta praevia.)

FIG. 8. Marginal placenta praevia. (*A*) Horizontal lateral view of uterus. The superior margin of the placenta is visible on the posterior wall of the uterus. (*B*) Upright lateral view of the pelvis. The fetal head is displaced anteriorly by the placenta which is located posteriorly. The distance between the fetal head and the gas shadow in the rectum represents the width of the placenta. (Verified.)

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filled amniotic sac protruding through the cervix occupies the position usually occupied by the head and may be confused with a centrally located placenta praevia. A rectal examination should readily differentiate between the two conditions.

7. A distended bladder (Fig. 5) or rectum may displace the fetal head posteriorly or anteriorly respectively and for these reasons the bladder and rectum should be emptied before examination. While a cystogram, with air or opaque medium (Fig. 6), and air or barium in the rectum (Fig. 8) may aid in the localization of the placenta by the abnormal width between the fetal head and the bladder or rectum, their use

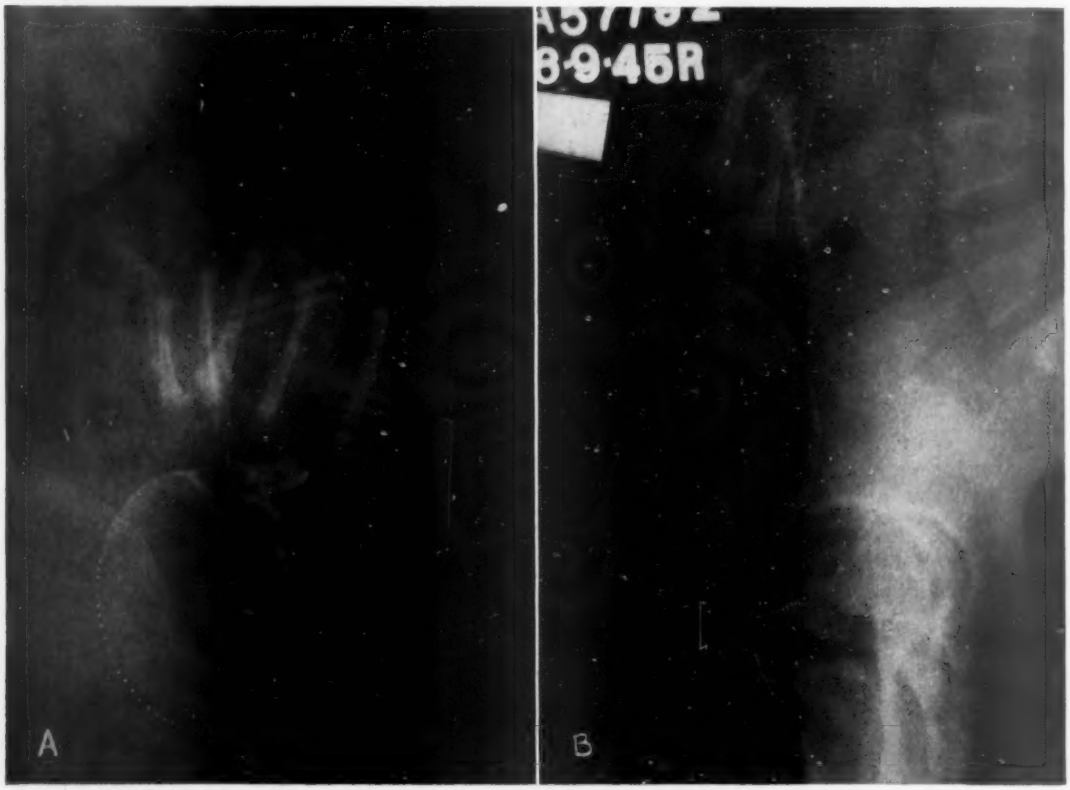

FIG. 7. Marginal placenta praevia. (*A*) Horizontal lateral view of uterus. The superior margin of the placenta is visible on the posterior wall of the uterus. (*B*) Upright lateral view of the pelvis. The fetal head is displaced anteriorly by the placenta which is located posteriorly. (Verified.)

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FIG. 9. Marginal placenta praevia. (*A*) Upright anteroposterior view of pelvis. The fetal head appears high and is displaced laterally. (*B*) Upright lateral view of pelvis. The fetal head is displaced anteriorly and does not engage. The placenta is located posteriorly and laterally. (Verified.)

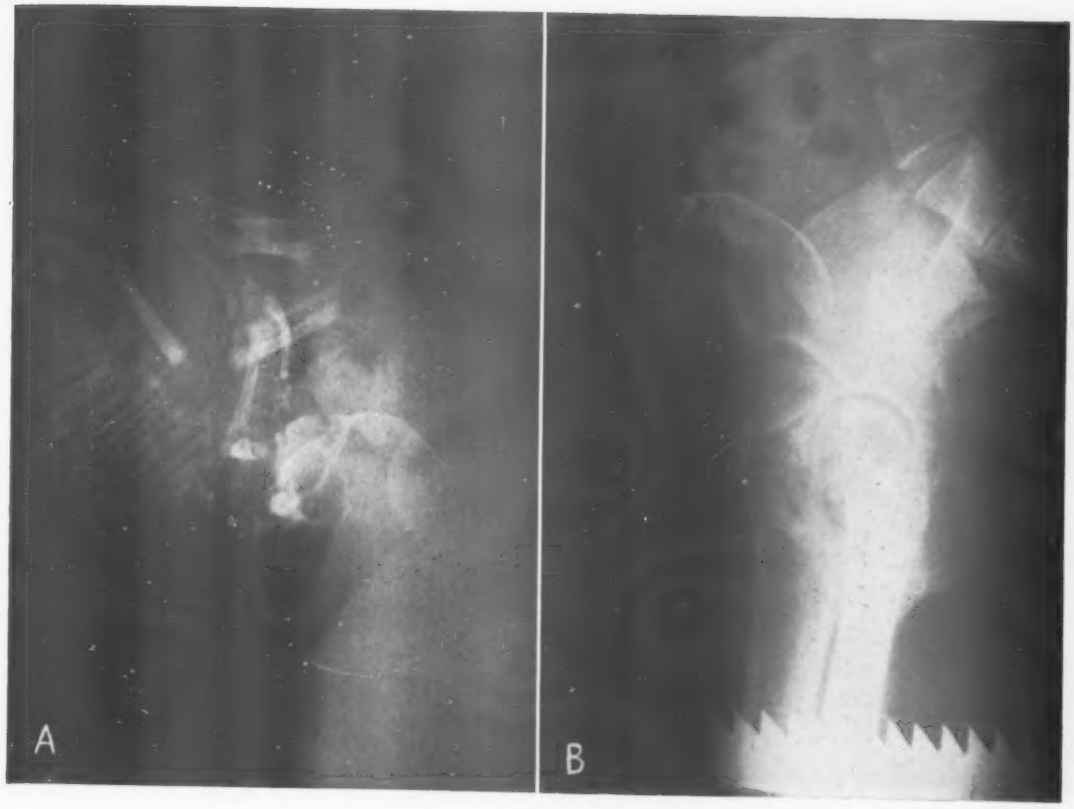

FIG. 8

FIG. 9

should be restricted to those cases examined in the upright position. Since the fetal head shifts when the patient is horizontal and may rise completely out of the pelvis, the normal distance between the fetal head and bladder and rectum may thus be interpreted as an abnormal finding attributed to a pelvic tumor.

Due to marked variation in size of placentas it is not safe to exclude a marginal placenta praevia by simple visualization of this structure in the body of the uterus. Upright films are necessary to determine the inferior margin of the placenta (Fig. 7 and 8).

We have found that the angle of inclination of the pelvic inlet has little or no effect on the centering of the fetal head. The fetal head has centered even in those multipara with pendulous abdomens and in primipara before so-called lightening has occurred. Were these patients examined in the upright position by the obstetricians, their observations would doubtless confirm the roentgen findings.

This exceedingly simple examination also eliminates the necessity of elaborate measurements of the fetal head and the pelvic inlet.

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CHONDROMA OF THE LARYNX

REPORT OF A CASE

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CHONDROMA of the larynx is rare. Moore[1] cited 53 cases from the world literature up to 1925, after excluding 9 other cases as unproved. By 1944, 32 more cases were reported, bringing the total number to 85. The literature on chondroma of the larynx was reviewed and the subject was comprehensively discussed in the papers by Moore in 1925 and again by McCall, Dupertuis and Gardiner[2] in 1944. Another detailed review would therefore be superfluous at this time. It is the purpose of this paper to add another case to the literature on this unusual condition.

CASE REPORT

The patient, a white male, aged fifty-five, native of the United States, was admitted to the New York Eye and Ear Infirmary on February 5, 1947, with complaint of persistent hoarseness for the past three years. In recent months he had difficulty in breathing.

Past medical and family history was essentially negative.

FIG. 1. Lateral view of larynx showing a partly calcified and ossified chondroma arising from the posterior wall of the larynx at the level of the cricoid.

Physical examination revealed an elderly white male not appearing ill. His voice was hoarse. Examination of the ears, nose and pharynx showed no significant pathologic findings. Laryngeal examination revealed a subglottic mass involving the left vocal cord, restricting its mobility and partly occluding the glottis on phonation. Examination of the lungs, heart and abdomen showed no abnormality.

FIG. 2. Anteroposterior tomogram of chondroma of the larynx arising at the level of the cricoid and growing upward to impinge on the laryngeal ventricle.

Blood pressure was 118/80. The clinical impression was left subglottic mass.

On February 6, 1947, roentgen examination of the larynx, including the lateral projection and anteroposterior tomography, revealed a large round soft tissue mass arising from the posterior wall of the upper trachea and cricoid and extending upward to impinge on the vocal cords causing partial obliteration of the airway. These findings suggested a calcifying and ossifying soft tissue tumor, probably an osteochondroma of the larynx (Fig. 1 and 2).

rounding structures. Speech is interfered with and the passage of air through the larynx is obstructed in varying degrees. Hoarseness, dyspnea and cough are common. Chondromas usually grow inward, obstructing the airway and impairing laryngeal function. Outward growing chondromas such as those from the outer surface of the body of the thyroid cartilage, manifest themselves as palpable tumors or produce pain and dysphagia. Superimposed

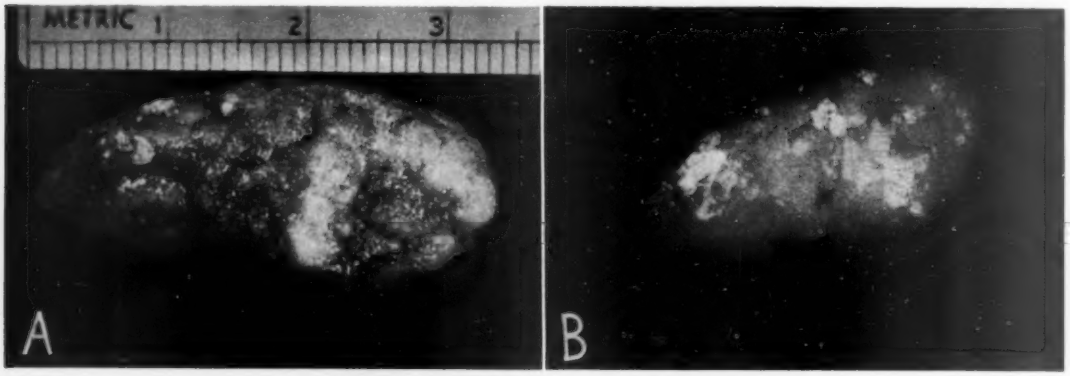

FIG. 3. (*A*) Surgical specimen of chondroma of the larynx, showing calcareous surface spots. (*B*) Roentgenogram of surgical specimen.

On February 6 a laryngeal fissure operation ws performed under local anesthesia. A large bone-hard mass covered by mucous membrane extended from the left wing of the thyroid cartilage, posteriorly, to the posterior signet of the cricoid and upper ring of the trachea It was dissected free and removed in toto (Fig. 3).

Postoperatively the patient's course was uneventful. He was discharged to his home on the fourth postoperative day.

Pathological Report. Microscopic section shows islands of hyaline cartilage separated by spaces with red blood cells. At some points in decalcified sections there is bone formation. This process is essentially a chondroma without evidence of malignant change.

Diagnosis. Chondroma of the larynx.

CRITERIA FOR DIAGNOSIS

Early diagnosis of chondroma of the larynx is difficult because it is slow growing and its symptoms and physical signs appear late. The symptoms arise from impairment of laryngeal function and pressure on surrespiratory infection may precipitate severe dyspnea and stridor in a patient whose symptoms had been mild. Emergency treatment with tracheotomy may then be required.

Laryngoscopy reveals a sessile or occasionally a pedunculated laryngeal mass whose mucosal blood vessels are prominent. Figi[3] stresses this feature as well as the hardness of the mass, as indicating the diagnosis of chondroma.

The most frequent sites of origin of chondroma are the posterior segment of the cricoid, the thyroid cartilage, the epiglottis, and the arytenoids.

Roentgenography is of increasing value in the diagnosis of laryngeal tumors. The lateral projection is helpful in profiling the extent of a tumor above and below the glottis. In recent years tomography (planigraphy) has been used increasingly for delineating laryngeal tumors at different depths in the anteroposterior projection. If

the tumor contains irregular calcium or bone deposits, the roentgenological diagnosis of calcifying or ossifying chondroma is made easy. Virchow[4] has stated, however, that the process of ossification in a chondroma requires twenty to thirty years. Of 30 cases reviewed by McCall *et al.*[2] since 1925, a mass or calcifying tumor was described in the roentgen report in each of 6 cases; in 3 the roentgen diagnosis was specifically chondroma.

Jackson and Jackson[5] stress the importance of direct laryngoscopy, palpation and biopsy. If a tumor is hard to palpation with a forceps a chondroma is suspected. The hardness of this tumor often makes excision of tissue for biopsy difficult.

DIFFERENTIAL DIAGNOSIS

Chondroma of the larynx, because of its rarity, is seldom suspected as the cause of laryngeal symptoms. Nonspecific laryngitis, polyps, tuberculosis and malignancy, being more common, are usually suspected as the cause of hoarseness, dyspnea and cough. The dyspnea associated with chondroma, when complicated by upper respiratory infection, may simulate bronchial and cardiac asthma, croup or aspirated foreign body.

PATHOGENESIS

Chondroma usually arises as localized overgrowth from laryngeal cartilage. It is usually not encapsulated and does not shell out. More rarely an encapsulated type of chondroma arises from cartilage rest cells. This type shells out easily, permitting the larynx to resume its normal shape and function. Chondromas often contain irregular calcifications and show characteristic calcareous surface spots (Fig. 3). Chondromas may ossify (osteochondroma); show myxomatous change (chondromyxoma); or undergo malignant degeneration (chondrosarcoma).

TREATMENT

The only effective treatment is surgical. Roentgen and radium therapy are of no value. With a small tumor direct laryngoscopic removal may be possible. Laryngeal fissure is usually the operation of choice. For extensive tumors, or if the tumor has recurred after laryngeal fissure operations, total laryngectomy may be necessary.

SUMMARY

A case of chondroma of the larynx has been reported. The diagnosis was based initially on a characteristic roentgenographic appearance of the tumor. Surgical findings and pathological examination confirmed the roentgenographic diagnosis.

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NEUTRON AND ROENTGEN-RAY EFFECTS ON PROTEIN CONTENT OF RAT INTESTINE

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STUDIES of the effects of penetrating radiation[4,5] on the chemical constituents of the cells of the crypts of Lieberkühn indicated a marked decrease in amounts of desoxyribonucleic acid, ribose nucleic acid, inorganic ash, and structural protein. The present investigations which extend these cytochemical studies indicate that changes in the concentration of total protein in the cells of the crypts of Lieberkühn and in total nitrogen in the intestinal mucosa also occur after irradiation with roentgen rays and with neutrons.

EXPERIMENTAL

White rats of the Brooklyn strain previously studied for nucleic acid changes following irradiation were used for the estimation of total protein. Ten rats received 600 r, roentgen radiation, 9 received 56 n, neutron radiation, 10 received 100 n, and 10 received 200 n. Each irradiated rat was paired with a control rat of the same weight. All were killed twenty-four hours after completion of irradiation. Sections of duodenum were taken from all the rats and were fixed in a 3:1 alcohol-acetic acid mixture.

Each duodenum from an irradiated animal was embedded together with the duodenum from a control animal of the same weight. Both tissues were sectioned at 3 microns with the same stroke of the microtome knife in order to have sections of similar thicknesses.

TYROSINE AND TRYPTOPHANE DETERMINATION

Pairs of successive sections from each block were mounted on opposite ends of a glass slide. One pair was completely treated with Millon reagent; the other, a control pair, was treated in a similar manner except for omission of treatment with NO_2; the latter served as "blanks."

The sections were mounted in glycerin, sealed with paraffin, and photographed according to the techniques described previously[3] with the ultraviolet microscope at 3654 Å. Since the spectrum of nucleic acid with its maximum at 2600 Å overlaps that of protein with its maximum at 2800 Å, and the absorption by protein is small when compared to that of nucleic acid, this makes it difficult or impossible to determine protein content by absorption at 2800 Å. Pollister and Ris[8] have shown that the maximum absorption of tyrosine and tryptophane, as constituents of protein, can be shifted from the region of 2800 Å to 3600 Å by treatment with the Millon reagents. For this reason, 3654 Å was chosen.

Photographs were taken of at least three different fields of the crypts of Lieberkühn in each of the four sections and of at least one from an area of the slide where no tissue was present to obtain the "background density." All photographs were taken on the same strip of film under identical conditions of light, exposure, and development. In the determination of the amount of absorption of the tyrosine and tryptophane portions of the protein in the tissue sections, density of the image on the film was determined by the use of a micro-densitometer.[4] At least thirty areas of each frame were read. The estimated value of the extinction coefficient of the tissue tyrosine and tryptophane was obtained by taking the average of the logarithmic values of the readings after correction for background and the blank.

A significant decrease in the tyrosine and tryptophane absorption at 3654 Å in the crypts of Lieberkühn was found twenty-four hours after the rats received 600 r.

The results (Table 1) show that the average extinction coefficient decreased in irradiated tissues and was 28.9 per cent less than that of the controls.

Six of the 9 animals which received a neutron dose of 56 n showed a decreased amount of absorption by tryrosine and tryptophane at 3654 Å; 1 showed no change; 2 showed an increase. The average decrease

TABLE I

EFFECT OF NEUTRON AND ROENTGEN IRRADIATION ON THE TYROSINE AND TRYPTOPHANE CONTENT OF THE CRYPTS OF LIEBERKÜHN

Irradiation	No. of Animals	Average Extinction Coefficient		Per Cent Decrease Extinction Coefficient
		Control	Irradiated	
600 r	10	0.368	0.262	28.8
56 n	9	0.373	0.340	8.8
100 n	10	0.454	0.342	24.7
200 n	10	0.356	0.208	41.6

in extinction coefficients (Table 1) in irradiated tissue was 9.0 per cent less than that of the controls.

A significant and marked decrease in absorption at 3654 Å, indicating decreased amounts of tyrosine and tryptophane, was found in the crypts of Lieberkühn twenty-four hours after the animals had received 100 n or 200 n. The decrease was consistent for all of the 20 irradiated rats. The average extinction coefficient decreased 24.6 per cent and 41.4 per cent respectively (Table 1) for those rats given 100 n and 200 n. The data indicate a greater decrease in tyrosine and in tryptophane content in those animals given 200 n than in those given either of the two smaller doses of neutron radiation.

ARGININE DETERMINATION

A modification of the Serra method[9] was used on pairs of tissues sectioned at 7 microns. After deparaffination, the sections were coated with a thin layer of collodion and the alpha-naphthol hypobromite reaction was carried out at near freezing temperatures by use of dry ice. The tissues were then mounted in glycerin and the coverslip sealed to the slide with paraffin. Visual examination of the section was made through a microscope equipped with a green filter.

Sections of duodenum showed a decrease in arginine content in 8 of the 10 animals given 100 n. In all of the 10 animals given 200 n there was a greater decrease than in those given 100 n. All of the crypts of Lieberkühn of the animals which had received 600 r showed a decrease.

NITROGEN

Fifty white rats, approximately 125 grams each, were divided equally into five groups of 10 animals and treated in the following manner:

Group 1—untreated controls—food ad lib.
Group 2—irradiated with 200 n—killed 24 hours after completion of irradiation.
Group 3—diet restricted for 24 hours to the amount of food eaten by group 2—killed at the end of this period.
Group 4—irradiated with 200 n—killed 48 hours after completion of irradiation.
Group 5—diet restricted for 48 hours to the amount of food eaten by group 4—killed at the end of this period.

All of the rats were decapitated. The entire small intestine from the stomach to the cecum was removed and opened longitudinally to expose the mucosa. The intestine was thoroughly washed in cold running water and placed in a heavy walled conical 50 milliliter centrifuge tube. A stainless steel spatula was forcibly applied with a crushing rotary motion to the intestine to free the mucosa from the muscularis. Three to five minutes was necessary to strip the mucosa from the muscularis. About 20 cc. of acetone was added to the macerate and the crushing procedure repeated. After the mucosa-free muscularis was discarded, the mucosa was washed three times with acetone. After each centrifugation, the super-

natant fluid containing acetone was discarded. The mucosa was dried for five hours at 40° C. Microchemical determinations for nitrogen[6] were made on the white, finely divided powder.

A significant decrease was found in the total dry weight of the mucosa from the irradiated animals which had been sacrificed twenty-four hours after irradiation and a greater decrease in those sacrificed forty-eight hours after irradiation (Table II). There was no significant change in the values found for nitrogen in the intestinal mucosa of those animals whose food had been restricted. Slight decrease in percentage of nitrogen was noted in animals which had received 200 n and had been killed twenty-four hours after irradiation; a somewhat greater decrease was found in those animals which had been killed forty-eight hours after irradiation (Table II). A reduction was found in the calculated total nitrogen of the mucosa of the small intestine of animals which had been irradiated while there was no reduction in the calculated total nitrogen in unirradiated animals whose food had been restricted (Table II).

TABLE II

NITROGEN CONTENT OF ACETONE-DRIED INTESTINAL MUCOSA FROM NEUTRON IRRADIATED AND FOOD RESTRICTED RATS

No. of Rats	Treatment	Average Nitrogen in Dry Mucosa	Average Dry Weight of Intestinal Mucosa	Calculated Amount N_2 in Dry Mucosa	Average Loss of N_2
		per cent	mg.	mg.	
10	Untreated	13.50	438	59.1	
10	Irradiated 200 n. Killed 24 hr. later	13.27	288	38.2	35%
10	Unirradiated. Diet restricted to amount of food consumed by group 2	13.25	442	58.6	
10	Irradiated 200 n. Killed 48 hr. later	12.93	123	15.9	73%
10	Unirradiated. Diet restricted to amount of food consumed by group 4	13.32	438	58.4	

DISCUSSION

The decrease in nucleic acid content was found to be dependent upon the dose as well as upon the length of time which had elapsed after irradiation.[4,5] The present results indicate that the decrease in total protein content of the cells of the crypts of Lieberkühn is also dependent upon the dose; the greater the amount of irradiation, the less total protein present. No timed series of the cytochemical determinations of protein was made, but the results of the timed series of total mucosal nitrogen determinations indicate that these changes are progressive.

Numerous nitrogenous substances other than protein are part of the total nitrogen in tissue, but the greater part of the nitrogen present is that of protein. Nucleic acid and protein have been shown to decrease after irradiation. Differences in the rate of decrease of these substances or of other nitrogenous materials may produce a change in percentage of nitrogen in the dry tissue even though there is no qualitative change in either the nucleic acid, protein, or other nitrogen bearing compounds. The very small change observed after irradiation in percentage of nitrogen may mean there is a difference in the ratio of these cellular constituents to one another.

Not only are there fewer cells after irradiation but presumably each cell contains less material. Cell size and nuclear size are considerably increased twenty-four hours after roentgen or neutron irradiation.[4,5] The decrease in the amount of total dried mucosa indicates, however, that there is

not only a decrease in total amount of protein, nucleic acid, etc., but that the existing substances in each cell are diluted still further by increased water concentration or by acetone soluble substances.

Irradiated animals have been found to eat less than non-irradiated control animals. In order to determine whether a reduction in the food intake may lead to a reduction in the amount of tissue protein, non-irradiated fasted animals were studied. Since no change was found in the amount of any of these substances after food restriction, the decrease in protein content of the intestinal mucosa cannot be explained by lowered food consumption and hence seems to be a phenomenon brought about by irradiation.

The dose of roentgen rays required to produce the same degree of change in the protein content in the cells of the crypts of Lieberkühn is about six times greater than that of neutrons. The decrease in protein twenty-four hours after irradiation with 600 r was found to be only slightly greater than the decrease after 100 n.

Some evidence of a close relationship between ribonucleic acid activity and protein synthesis has been offered.[1] A parallel relationship was found in this study, where a decreased amount of nucleic acid was found to be associated with decreased amounts of protein after irradiation. These results do not indicate whether the synthesis of nucleic acid and protein are interdependent.

Denaturation of proteins,[10] changes in the phenol groups of tyrosine and tryptophane,[11,12] inactivation of enzymes,[2] and depolymerization of nucleic acid[13,14] when pure solutions were irradiated with roentgen rays have been described. Mazia[7] has shown that pepsin in the dry form is inactivated with small doses of roentgen radiation. It has been shown that these substances in solution can be protected[11] by the addition, prior to irradiation, of numerous other compounds. Whether the decrease in nucleic acid and protein content represent a direct effect of irradiation or an indirect effect is not known. In a chain of interdependent reactions, however, it would seem that the nucleic acid and protein changes are closer to the primary effect than the strictly morphological changes previously reported in the literature.

SUMMARY

A reduction in the amounts of tyrosine, tryptophane, arginine and nitrogen, which are important constituents of protein, is evidence of a reduction in the amount of protein after neutron and roentgen irradiation.

Cytochemical determinations of the total protein content in tissue sections was estimated through the study of the tyrosine and tryptophane ultraviolet absorption at the wavelength 3654 Å. The tyrosine and tryptophane content was markedly decreased in the crypts of Lieberkühn twenty-four hours after 600 r total body roentgen irradiation, but there was no significant change in the tyrosine and tryptophane content of the crypts of Lieberkühn twenty-four hours after 56 n total body neutron irradiation. A considerable decrease in the tyrosine and tryptophane content of the crypts of Lieberkühn was found twenty-four hours after 100 n or 200 n total body irradiation respectively.

The arginine content was found to decrease twenty-four hours after 600 r or 200 n total body irradiation.

The percentage of nitrogen was not markedly changed in the acetone dried intestinal mucosa in irradiated or non-irradiated fasted animals. The amount of acetone-dried intestinal mucosa was significantly changed after twenty-four and forty-eight hours in those animals irradiated with 200 n, but was not changed in those non-irradiated animals whose food had been restricted for the same periods of time. The calculated total nitrogen for the acetone-dried intestinal mucosa was found to be greatly decreased twenty-four and forty-eight hours respectively after 200 n neutron radiation, but there was no sig-

nificant reduction in non-irradiated fasted animals.

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NEUTRON EFFECTS ON ALKALINE PHOSPHATASE OF RAT INTESTINE

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A RAPID and significant change was reported[2,3,9] in the concentration of ribonucleic acid, desoxyribonucleic acid, protein and ash in the crypts of Lieberkühn of the rat as a result of neutron and roentgen irradiation. These changes in nucleic acid and protein suggested a need for further studies of the derangement of nucleoprotein metabolism in irradiated animals.

Krugelis[6,7] showed in cytochemical determinations that alkaline phosphatase was closely associated with nucleic acid. Since alkaline phosphatase is involved in the metabolism of phosphoesters, any change in the activity of phosphatase after irradiation would indicate that there might be changes in the metabolism of phosphoric-acid-bearing substances, especially of ribonucleic acid and desoxyribonucleic acid.

EXPERIMENTAL

Ten male albino rats of the Brooklyn strain, weighing 150 grams each, were irradiated with a neutron dose of 200 n. Each irradiated animal was paired with an unirradiated control animal of the same weight. Twenty-four hours after irradiation the irradiated and the control rats were decapitated and sections of duodenum were taken and fixed in cold 95 per cent alcohol. Each of the sections of duodenum from an irradiated animal was embedded in paraffin with the section from an unirradiated control animal. Both tissues were sectioned at 6 μ with the same stroke of the microtome knife.

Alkaline phosphatase was determined according to the Gomori method[4] on pairs of successive sections from each block incubated in different substrates. The phosphomonoesters used for substrates were beta glycerophosphate, yeast adenylic acid, and yeast guanylic acid; the phosphodiesters used were enzyme depolymerized desoxyribonucleic acid[6], ribonucleic acid, and diphenyl phosphate. The sections were incubated from one-half to twenty-four hours in solutions of the various substrates containing magnesium and calcium salts until the optimal reaction was obtained for each substrate at pH 9.0 with veronal buffer and pH 7.0 with acetate buffer. Since the controls and irradiated sections were of the same thickness, mounted on the same slide and treated alike, any difference in the amount of cobalt sulfide would be due to differences in the amount of phosphatase enzyme or its activity. Sections were also incubated at pH 9.0 and 7.0 in a medium that contained no phosphoester and then treated with the cobalt and ammonium sulfide

Essentially the same phosphomonoesterase reaction was obtained at pH 9.0 whether the beta-glycerophosphate, adenylic acid or guanylic acid was used, but consistent differences were noted between control and experimental tissues. The cobalt sulfide blanks showed no apparent "staining," while control tissues incubated in a medium containing a phosphoester showed slight but definite "staining" in the cells of the crypts and somewhat greater "staining" in their nucleoli. Sections from irradiated animals with a phosphomonoester substrate showed deeper "staining" in the cytoplasm, nucleus and especially in the nucleolus, than those from controls. The reaction in the crypts of Lieberkühn was not as strong as the reaction in the cytoplasm, nucleus and nucleolus of the villi.

With the phosphodiesters diphenyl phosphate, desoxyribonucleic acid or ribonucleic acid at pH 9.0 the activity in the nucleus and nucleolus of the cells of the crypts of Lieberkühn in tissues from irradiated ani-

mals was stronger than the very weak reaction found in the control sections. There was also a stronger cytoplasmic reaction in sections from irradiated animals than from controls, but the "staining" was uneven. With the phosphomonesters the reaction in the crypts of the control sections was similar to the reaction obtained in the control specimens.

With all of these substrates no difference could be found in the cells of the villi between the controls and experimental animals.

At pH 7.0 no differences were observed between tissues from irradiated and from control animals. No differences were observed in size or intensity of "staining" of the Golgi apparatus which reacts at this pH.

DISCUSSION

Radioactive phosphorus turnover has been shown by Scott[10] to be decreased after roentgen and neutron irradiation. Hevesy[5] showed that the turnover of desoxyribonucleic acid and phosphatide phosphorus of the nucleus was also decreased. Scott's results give no indication that the catabolic processes involving nucleic acid were altered but only that the amount of phosphorus utilized was decreased. A considerable decrease in the number of cells of the crypts of Lieberkühn had been found twenty-four hours after neutron irradiation of rats[3] and also in the amount of nucleic acid, protein and inorganic constituents. The cells which remained were enlarged and contained larger nuclei and nucleoli. The activity of the alkaline phosphatase in such cells now has been found to be increased. In the light of the findings that nucleic acid was reduced in amount after irradiation, it would seem that the reduced turnover of radioactive phosphorus was related to the reduced synthesis of nucleic acid or to the decrease in number of living cells.

The complex phosphorus-bearing products of cell destruction and cell injury, which may be the result of irradiation, are probably acted upon by alkaline phosphatase and are reduced to simpler substances. The progressive decrease in nucleic acid for the first few days after irradiation together with the loss of phosphorus would indicate that the increased phosphatase activity is dependent upon such cell damage and is of a catabolic nature. This increase in activity would result in a loss of phosphorus from the cell[1] and may be the cause of the increase in inorganic phosphorus found in blood after irradiation.[11] At pH 7.0, which is more nearly that of the pH of living tissue, however, the phosphatase reactions in control and irradiated tissues were found to be similar.

An increase in phosphatase activity has been noted by Wachstein[11] in livers of rats after dietary protein depletion. It has been shown in previous studies[3] that there is a protein depletion after roentgen and neutron irradiation. In the present study a decrease in phosphatase activity was found. While different techniques were used to produce a protein depleted condition, the results are similar. The relation, if any, however, between protein depletion and the increased phosphatase activity is not evident.

SUMMARY

The effect of neutron irradiation on the alkaline phosphatase of the rat duodenum was investigated. An increase in alkaline phosphatase activity at pH 9.0 was found in the nuclei and nucleoli in the cells of the crypts of Lieberkühn when beta-glycerophosphate, desoxyribonucleic acid, ribonucleic acid, adenylic acid, guanylic acid, or diphenyl phosphate was used as the source of organic phosphorus.

No difference could be observed between the phosphatase activity of the cells in the villi of the duodenum from irradiated and from non-irradiated animals.

No difference in phosphatase activity at pH 7.0 was noted in any of the parts of the duodenum of irradiated and non-irradiated animals with any of these substrates.

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DEPARTMENT OF TECHNIQUE

Department Editor: Robert B. Taft, M.D., B.S., M.A., 103 Rutledge Ave.
Charleston, S. C.

TRAINING OF THE BLIND FOR DARK ROOM TECHNICIANS

By SHERWOOD MOORE, M.D.
Director of the Mallinckrodt Institute of Radiology
ST. LOUIS, MISSOURI

THE very best possible roentgen technique is only as good as the dark room processing of the films. The best roentgen technician can fail because of poor dark room technique. The methods of solving this problem have been many and varied. Every combination of personnel and method has been tried with almost universally poor results.

Having heard of a radiologist who employed a blind dark room technician a year or so ago, I reflected over that matter and recalled the times when I had to perform my own dark room work in emergencies. Frequently I did not take the trouble to turn on the red lights in the dark room, relying on the sense of feel and the time clock to process the negatives. Putting these two points together it seemed a desirable experiment to attempt the employment of blind individuals for this function of a roentgen department.

The local Bureau for the Blind was consulted and showed immediate interest. It placed three blind men in the laboratory in order to see if they could master the procedure. These three men were instructed by the senior roentgen-ray technician for a period of about a month. By that time they had acquired manual proficiency which justified attempting to use them under actual working conditions. A month's experience satisfied me that, if permission could be secured, they could be put on as permanent personnel. This has continued for the past seven months with extremely satisfactory results.

A word of explanation as to the conditions under which these blind technicians work. There are two dark rooms, one of the conventional type and the other finished with the automatic processing machine. The conventional dark room was designed for two dark room technicians, one to load and unload cassettes and hangers with his hands dry and the other to be at the solution tanks. They, of course, alternated. With the automatic machine one blind technician carries the entire load. In an eight hour day these three processed from 400 to 600 films per day.

As to results, the technical roentgenographic quality is of a high order. The roentgen-ray technicians are satisfied since their efforts are not nullified by careless dark room technique. They do not have to give any help in the dark room and indeed do not have to go into it. Their services are more important and in greater need outside of the dark room than they ever would be inside. Best of all, the blind men are happy. As one of them commented, "This is far better than making brooms." The success of this experiment was so satisfactory that it stimulated my interest in helping the blind and, second, in contributing something to other roentgenologists who, if they have sufficient dark room work, would find themselves much happier with this part of the activities cared for by a blind individual. These two considerations are the chief ones for bringing the matter to the notice of roentgenologists. Again, there is the hope that the

film industry and commercial photographic laboratories will take up this form of employment for those handicapped by loss of sight. Several outstanding radiologists were communicated with in the hope of arousing their interest. This met with some quite favorable reactions. I found out that the idea was not in the least original with me, that several roentgenologists had employed the blind with considerable success. Dr. Howard P. Doub, Editor of *Radiology*, called my attention to an editorial in that journal on this subject several years ago.

In the correspondence a good many objections to the employment of the blind were brought up. These are about as follows: Fear that the blind person would need considerable help, danger of transit to and from work, existing satisfactory methods of conducting the dark room, a volume of work too small to justify a dark room technician. Other objections may develop in the future. The experience so far leads to the belief that properly selected and trained individuals would eliminate all objections so far advanced except possibly that of small volume of work.

In the Mallinckrodt Institute of Radiology films are processed on the "time and temperature" method with thermostatic controlled solutions and the photographic method of film identification. These blind employees have no difficulty with any phase of the work even though with thermostatic control temperatures will vary, but the blind man can make a time correction with a Braille thermometer. The blind can vary time of development even where laboratories have to use ice for cooling. They keep the solutions at the correct level and will doubtless manage the "replenisher" system which is in process of being installed. Preparation of the chemical solutions does have to be done by the roentgen-ray technicians.

Additional advantages to employing the blind are that the roentgen-ray technicians are freed from the monotony and fatigue of dark room work, where their superior abilities are more or less wasted.

Success in employing blind dark room technicians depends upon the selection of suitable individuals who should be thoroughly trained in advance of embarking on the actual work. There are certain limitations in the selection of individuals for training. They should be able to go and come without aid and find their way about a roentgen department without help. They should be temperamentally equipped for

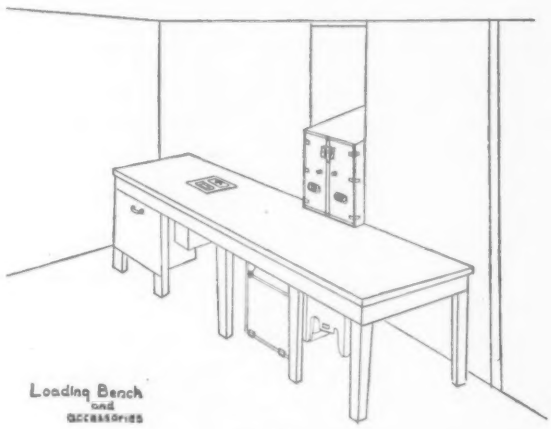

Fig. 1

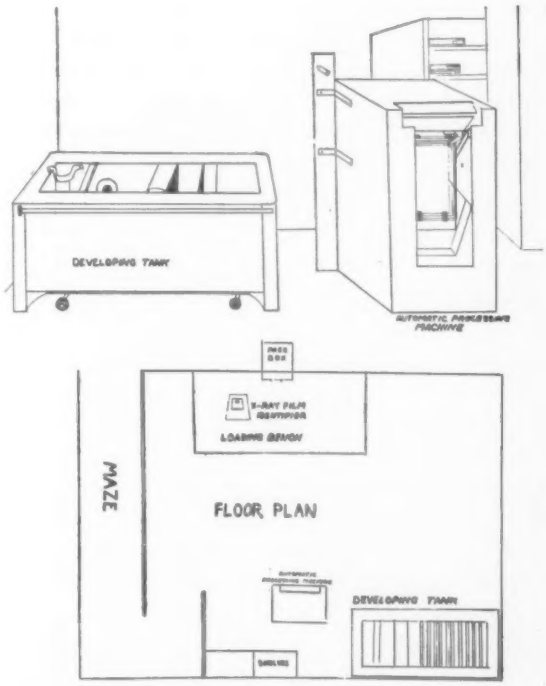

Fig. 2

the training and the work. They should be given an aptitude test. They should be young enough to acquire, if they do not already possess it, manual dexterity. Either men or women can be trained (one girl has been trained). They should be conscientious and honest. The selection of individuals to be trained should be left to the state or federal agencies for the blind. These agencies have the facilities for giving the aptitude tests and find persons with the proper background and can help in the placement once the blind person has been trained.

Our success coupled with some applications for training were sufficient that a pilot dark room was set up with the necessary appliances for training. Training was restricted to those referred by the agencies mentioned above. (Figures 1 and 2 are black line drawings illustrating this pilot dark room.)

The trainees are instructed by the senior technician until they acquire such speed and dexterity that they can be entrusted with actual dark room work. The pilot dark room naturally is "dry." They are drilled with loading and unloading films from cassettes and loading and unloading hangers and hangettes until time studies measure their efficiency. When the speed of operation approaches that of a fully trained technician the training is continued under actual working conditions. Length of training will vary with the individual. Parenthetically, the blind employees can unload and load and place a film in solution as speedily as can a technician with sight. Training requires about two weeks in the pilot dark room after which they are transferred to the actual dark room to do the daily work.

The Federal Security Agency, Office of Vocational Rehabilitation, and the Illinois Division of Vocational Rehabilitation and a representative from the Bureau for the Blind have investigated this training program and expressed the view that it has possibilities for a good livelihood for the blind. It is not and should not be a method of getting cheap help.

There is full realization that there are limited opportunities in departments of roentgenology. However, as a step toward training the blind for the film industry and commercial photography this training project may open a door for large opportunities.

In departments of roentgenology, if the photographic method of film identification is used, this can be easily done by the blind. Other methods of film identification present a real difficulty which, however, is not insurmountable. In the training plan for the blind those who learn to write before they have lost their vision are trained to write the name and date on a film before it is processed. Even if in a department of roentgenology film identification is a bar to the blind, their employment is well worth while to load and unload cassettes, hangers and hangettes.

The roentgen-ray industry has given whole hearted and generous support. They have amply supplied outdated film for "dry" training, hangers, cassettes and permission to reconstruct the loading end of the automatic machine. Because of this, the expense of training is reduced to a minimum. The only expense to the representative bureaus is that of travel, subsistence and housing for the trainees plus compensation for the roentgen-ray technician who gives the training on his own time.

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Editorial Board: A. C. Christie, E. H. Skinner, Lauriston S. Taylor.

Advisory Board for Pathology: Eugene L. Opie.

Fifty-first Annual Meeting: Jefferson Hotel, St. Louis, Mo., Sept. 26–29, 1950.

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President: William S. MacComb, New York, N. Y.; *President-Elect:* Leland R. Cowan, Salt Lake City, Utah; *1st Vice-President:* James A. Corscaden, New York, N. Y.; *2nd Vice-President:* Douglas J. Roberts, Hartford, Conn.; *Secretary:* Hugh F. Hare, 605 Commonwealth Ave., Boston, Mass.; *Treasurer:* Howard B. Hunt, University Hospital, Omaha, Nebr.

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Exhibit Committee: Robert E. Fricke, Chairman, Rochester, Minn., William Harris, New York, N. Y., Milton Friedman, New York, N. Y.

Thirty-second Annual Meeting: Biltmore Hotel, New York, N. Y., May 25–26, 1950.

EDITORIALS

THE ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

THE Fiftieth Annual Meeting of the American Roentgen Ray Society held in Cincinnati, Ohio, October 4 to 7, 1949, was outstanding in many respects. Held under the presidency of Dr. U. V. Portmann of Cleveland, Ohio, the Scientific Program was among the best that has been presented before the Society. On the opening day, following the call to order by the outgoing President, Dr. Lawrence Reynolds, and his presentation of the scientific awards, the Society was welcomed to Cincinnati by Dr. Raymond Walter, President of the University of Cincinnati, and in a very interesting address he sketched briefly the history of Cincinnati and the important part the city has played in the development of the Midwest.

The American Roentgen Ray Society always welcomes an opportunity to return to Cincinnati for its annual meeting; because it is so centrally located, it is accessible to all parts of the country and the facilities of the meeting place, the Netherland Plaza, are unsurpassed.

Dr. Portmann, after his installation as President, gave a very interesting address on "Therapeutic Radiology as a Specialty." In this he discussed the various aspects of therapeutic radiology, its present position within the framework of the radiological departments of hospitals and universities, and suggested some modification of the position of therapeutic radiology in the organization of radiological departments. Dr. Portmann's address will be published in full in a forthcoming issue of the JOURNAL.

Dr. Portmann and his Program Committee had arranged a splendid program which was thoroughly enjoyed and from which all the members and guests profited. It is not necessary here to mention the various papers given for they will be published in future issues of the JOURNAL.

One of the highlights of the meeting was the Caldwell Lecture which occurred on Wednesday evening. The lecture was given by Dr. Stanley P. Reimann, Director of the Research Institute, Lankenau Hospital Philadelphia, and he discussed "The Differentiation of Cells and Tissues." In his brilliant talk Dr. Reimann called attention to the importance of the differentiation of cells and their concomitant and subsequent organization into tissues, parts and organs, and he stressed the fact that this is the very essence of a large part of biology and medicine and applications of such differentiation ramify into every nook and cranny of our daily thinking and work. These processes, he said, made us and our patients what we are; in fact, make all living things what they are. There has perhaps never been given before the Society a more fundamental and thought-provoking dissertation than that of Dr. Reimann.

This year's Scientific Exhibit may be favorably compared with the best at any of the previous meetings of the Society. To Dr. G. Allen Good and the members of his Scientific Exhibit Committee the Society owes its thanks. The Scientific Exhibit itself alone was worth a trip to Cincinnati and many of the scientific exhibits amplified and extended papers that were given in the scientific program. Elsewhere in this number of the JOURNAL is given a detailed account of the scientific exhibits shown and the awards given.

The Commercial Exhibits this year may also be accounted among the best that the Society has had and the Program Committee had quite properly set aside certain hours during the day for the members and guests to visit both the Scientific and Commercial Exhibits. These two exhibits are features of the annual meetings which are of such importance as to warrant this setting aside of specific hours for their inspection.

The day preceding the opening meeting was given over to the Annual Golf Tournament which was held at the Kenwood Country Club through the kindness and arrangement of the Local Committee. Competition for the Willis F. Manges Trophy was participated in by one of the largest groups of players of any tournament within recent years and the dinner following the golf tournament was perhaps the largest of any, certainly in a number of years. To the Chairman, Dr. Harold G. Reineke and his Local Committee, the Society expresses its very great appreciation not only for the arrangements for the annual golf tournament but for all of the other social functions, as well as the arrangements having to do with the various phases of the scientific program.

The annual banquet which was held on Thursday evening was as usual one of the highlights of the social activities of this year's meeting. Approximately five hundred attended the banquet which was held in the beautiful Pavillon Caprice. Following the banquet the Local Committee had arranged a splendid floor show after which there was dancing.

At the meeting, the Ladies Committee, with Mrs. Harold G. Reineke as Chairman, had arranged a splendid program for the ladies, including interesting trips to the various places of historical interest and to the splendid art museums in Cincinnati.

The success of the meeting was brought about not only by the splendid planning of President Portmann and his Program Committee and the Local Committees on Arrangements, but also by the Netherland Plaza and its personnel in cooperation with the Convention Bureau by seeing that those attending were properly and pleasantly housed, and as might have been anticipated the meeting in Cincinnati proved to be an excellent one in every respect.

ARIAL WELLINGTON GEORGE
1882-1948

DR. ARIAL WELLINGTON GEORGE, one of the nation's pioneer radiologists whose research in roentgen diagnosis of diseases of the gastrointestinal tract, gallbladder and spine had aided humanity throughout the world, died on December 24, 1948. Dr. George was born in Yonkers, New York, in 1882. He prepared for Tufts College at Tilton Academy and was graduated from the Tufts Medical School in 1906. Dr. George lived in Wellesley Hills and maintained an office in Boston for thirty-five years and had served in the clinical as well as the research field.

Arial Wellington George

In recognition of his application of the roentgen ray to medical diagnosis, the British Royal Society of Medicine selected him, in 1923, as one of the first Americans to receive the Sir James MacKenzie-Davidson Memorial Medal. Three years later he received an honorary Doctor of Science degree from Tufts College and an honorary Diploma in Medical Radiology and Electrology from the University of Cambridge, England.

He was a former teacher at the Tufts and the Harvard Medical Schools and he was the author of several books including "Surgical Lesions of the Gastro-intestinal Tract," "The Vertebrae" and "The Gall Bladder." He was also the author of many articles in scientific and medical publications.

At the time of his death, Dr. George was radiologist at the Brooks Hospital, Brookline, the Glover Memorial Hospital, Needham, and at the Massachusetts Institute of Technology Infirmary, and also a consultant at the Attleboro, the Cape Cod and the Lynn hospitals. He had formerly served as a consultant radiologist at the Boston Dispensary, the Boston City Hospital the Children's Hospital, the New England Hospital for Women and Children and the Forsyth Dental School.

Dr. George was a member of the American Roentgen Ray Society, a past chancellor of the American College of Radiology, a member of the Radiological Society of North America, the New England Roentgen Ray Society and the American Medical Association.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

MEETINGS OF ROENTGEN SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. Barton R. Young, 3401 North Broad St., Philadelphia, Pa. Annual meeting: Jefferson Hotel, St. Louis, Mo., Sept. 26–29, 1950.

American Radium Society
Secretary, Dr. H. F. Hare, 605 Commonwealth Ave., Boston, Mass. Annual meeting: Biltmore Hotel, New York, N. Y., May 25–26, 1950.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: Cleveland Auditorium and Statler Hotel, Cleveland, Ohio, Dec. 4–9, 1949.

American College of Radiology
Executive Secretary, William C. Stronach, 20 N. Wacker Drive, Chicago 6. Annual meeting: 1950, to be announced.

Section on Radiology, American Medical Association
Secretary, Dr. Paul C. Hodges, 950 E. 59th St., Chicago, Ill. Annual Meeting: 1950, to be announced.

Alabama Radiological Society
Secretary, Dr. W. W. Anderson, Tuscaloosa, Ala. Meets time and place Alabama State Medical Association.

Arizona Association of Pathologists and Radiologists
Secretary, Dr. R. Lee Foster, 507 Professional Bldg., Phoenix, Ariz. Two regular meetings a year. The annual meeting at time and place of State Medical Association and interim meeting six months later.

Arkansas Radiological Society
Secretary, Dr. Fred Hames, 511 National Bldg., Pine Bluff, Ark. Meets every three months and also at time and place of State Medical Association.

Atlanta Radiological Society
Secretary, D. W. W. Bryan, 490 Peachtree St., N.E., Atlanta, Ga. Meets monthly, except during three summer months, on second Friday evening.

Brooklyn Roentgen Ray Society
Secretary, Dr. J. J. Daversa, 345 75th St., Brooklyn, N. Y. Meets monthly fourth Tuesday, Oct. through April.

Buffalo Radiological Society
Secretary, Dr. Mario C. Gian, 610 Niagara St., Buffalo, N. Y. Meets second Monday evening each month, October to May inclusive.

Central New York Roentgen Ray Society
Secretary, Dr. Dwight V. Needham, 608 E. Genesee St., Syracuse N. Y. Meets January, May, November.

Central Ohio Radiological Society
Secretary, Dr. Paul D. Meyer, Grant Hospital, Columbus, Ohio. Meets at 6:30 P.M. on second Thursday of October, December, February, April, and June at Seneca Hotel, Columbus, Ohio.

Chicago Roentgen Society
Secretary, Dr. John H. Gilmore, 720 N. Michigan Ave., Chicago 11, Ill. Meets second Thursday of each month October to April inclusive at the University Club.

Cincinnati Radiological Society
Secretary, Dr. Eugene L. Saenger, 735 Doctors Bldg., Cincinnati 2, Ohio. Meets last Monday of each month, September to May, inclusive.

Cleveland Radiological Society
Secretary, Dr. J. R. Hannan, Cleveland Clinic, Cleveland 6, Ohio. Meetings at 6:30 P.M. on fourth Monday of each month from October to April.

Colorado Radiological Society
Secretary, Dr. Paul E. RePass, 306 Republic Bldg., Denver 2, Colo. Meets third Friday of each month at University of Colorado Medical Center or at Denver Athletic Club.

Connecticut Valley Radiologic Society
Secretary, Dr. E. W. Godfrey, 1676 Boulevard, West Hartford, Conn. Meets second Friday Oct. and April.

Dallas-Fort Worth Roentgen Study Club
Secretary, Dr. X. R. Hyde, Medical Arts Bldg., Fort Worth, Texas. Meets in Dallas on odd months and in Fort Worth on even months, on third Monday, 7:30 P.M.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. W. G. Belanger, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

East Bay Roentgen Society
Secretary, Dr. Dan Tucker, 434-30th St., Oakland 9, Calif. Meets first Thursday each month at Peralta Hospital, Oakland.

Florida Radiological Society
Secretary, Dr. F. K. Hurt, Riverside Hospital, Jacksonville, Fla. Meets twice annually, in the spring with the annual State Society meeting, and in the fall.

Georgia Radiological Society
Secretary, Dr. Robert Drane, DeRenne Apartments, Savannah, Ga. Meets in mid-winter and at annual meeting of Medical Association of Georgia in the spring.

Houston X-ray Club
Secretary, Dr. Curtis H. Burge, 3020 San Jacinto St. Houston 4, Texas. Meets fourth Monday each month.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Illinois Radiological Society
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meets three times a year.

Indiana Roentgen Society
Secretary, Dr. William M. Loehr, 712 Hume-Mansur Bldg., Indianapolis 4. Meets second Sunday in May.

Iowa X-Ray Club
Secretary, Dr. Arthur W. Erskine, 326 Higley Bldg., Cedar Rapids, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. Special meetings by announcement.

Kansas Radiological Society
Secretary, Dr. Anthony F. Rossitto, Wichita Hospital, Wichita, Kan. Meets annually with State Medical Society.

Kentucky Radiological Society
Secretary, Dr. W. C. Martin, 321 W. Broadway, Louisville. Meets annually in Louisville on first Saturday in Apr.

Kings County Radiological Society
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Los Angeles Radiological Society
Secretary, Dr. Wybren Hiemstra, 1414 S. Hope St., Los Angeles 15, Calif. Meets second Wednesday each month at Los Angeles County Medical Assn. Building.

Louisiana Radiological Society
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

Louisville Radiological Society
Secretary, Dr. E. L. Pirkey, Louisville General Hospital, Louisville 2, Ky. Meets monthly on second Friday at Louisville General Hospital.

* Secretaries of societies are requested to send timely information promptly to the Editor.

Michigan Association of Roentgenologists
Secretary, Dr. R. D. McDuff, 220 Genesee Bank Bldg., Flint 3, Mich.

Milwaukee Roentgen Ray Society
Secretary, Dr. T. J. Pfeffer, 839 N. Marshall St., Milwaukee 2, Wis. Meets monthly on second Monday at University Club.

Minnesota Radiological Society
Secretary, Dr. Chauncey N. Borman, 802 Medical Arts Bldg., Minneapolis 2, Minn. Two meetings yearly, one at time of Minnesota State Medical Association the other in the fall.

Nebraska Radiological Society
Secretary, Dr. Ralph C. Moore, Nebraska Methodist Hospital, Omaha 3, Nebr. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

New England Roentgen Ray Society
Secretary, Dr. George Levene, Massachusetts Memorial Hospitals, Boston, Mass. Meets monthly on third Friday, Harvard Club.

New Hampshire Roentgen Ray Society
Secretary, Dr. A. C. Johnston, Elliott Community Hospital, Keene, N. H. Meets four to six times yearly.

New York Roentgen Society
Secretary, Dr. F. H. Ghiselin, 111 East 76th St., New York 21, N. Y. Meets monthly on third Monday, New York Academy of Medicine, at 8:30 P.M.

North Carolina Radiological Society
Secretary, Dr. J. E. Hemphill, 1420 E. Fifth St., Charlotte 4, N. C. Meets in May and October.

North Dakota Radiological Society
Secretary, Dr. C. O. Heilman, 807 Broadway, Fargo. Meetings held by announcement.

Northern California Radiological Club
Secretary, Dr. C. E. Grayson, Medico-Dental Bldg., Sacramento 14, Calif. Meets at dinner last Monday, every second month, except June, July and August.

Ohio State Radiological Society
Secretary, Dr. E. C. Elsey, 927 Carew Tower, Cincinnati 2, Ohio.

Oklahoma State Radiological Society
Secretary, Dr. W. E. Brown, Tulsa, Okla. Three regular meetings annually.

Oregon Radiological Society
Secretary, Dr. Boyd Isenhart, 214 Medical Dental Bldg., Portland 5, Oregon. Meets monthly 2nd Wednesday, 8:00 P.M., Library of University of Oregon Medical School.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets first Tuesday of each month.

Pacific Northwest Radiological Society
Secretary, Dr. S. J. Hawley, 1320 Madison St., Seattle 4, Wash. Meets annually in May.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. J. M. Converse, 416 Pine St., Williamsport. Annual Meeting: May 20 and 21, 1949, Bedford Springs Hotel, Bedford, Pa.

Philadelphia Roentgen Ray Society
Secretary, Dr. G. P. Keefer, 1930 Chestnut St. Meets first Thursday each month October to May, at 8:00 P.M., in Thomson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. R. P. Meader, 4002 Jenkins Arcade Pittsburgh 22, Pa. Meets 6:30 P.M. at Webster Hall Hotel on second Wednesday each month, October to May inclusive.

Queens Roentgen Ray Society
Secretary, Dr. J. E. Goldstein, 88-29 163rd St., Jamaica 3, N. Y. Meets fourth Monday of each month except during the summer.

Radiological Section, Baltimore Medical Society
Secretary, Dr. Harry A. Miller, 2452 Eutaw Place, Baltimore. Meets third Tuesday each month, September to May.

Radiological Section, Connecticut Medical Society
Secretary, Dr. Fred Zaff, 135 Whitney Ave., New Haven, Conn. Meets bimonthly on second Wednesday.

Radiological Section, District of Columbia Medical Society
Secretary, Dr. K. C. Corley, 1835 Eye St., N. W., Washington, D. C. Meets Medical Society Auditorium, third Thursday, January, March, May, October at 8:00 P.M.

Radiological Section, Southern Medical Association
Secretary, Dr. Roy G. Giles, Temple, Texas.

Radiological Society of New Jersey
Secretary, Dr. Benjamin Copleman, 280 Hobart St., Perth Amboy, N. J. Meets annually at Atlantic City at time of State Medical Society and mid-winter at Elizabeth, N. J.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Ralph E. Alexander, 101 Medical Arts Bldg. Meets monthly on third Monday from October to May, inclusive, 8 P.M. at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. Maurice D. Frazer, 1037 Stuart Bldg., Lincoln, Nebr. Meets Shirley-Savoy Hotel, Denver, Colo. August 18, 19, 20, 1949.

St. Louis Society of Radiologists
Secretary, Dr. C. J. Nolan, 737 University Club Bldg., St. Louis 3, Mo. Meets fourth Wednesday each month, except June, July, August, and September.

San Diego Roentgen Society
Secretary, Dr. R. F. Niehaus, 1831 Fourth Ave., San Diego, Calif. Meets monthly, first Wednesday at dinner.

Section on Radiology, California Medical Association
Secretary, Dr. D. R. MacColl, 2007 Wilshire Blvd., Los Angeles 5, Calif.

Section on Radiology, Illinois State Medical Society
Secretary, Dr. Harold L. Shinall, St. Joseph's Hospital, Bloomington, Ill.

Shreveport Radiological Club
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

South Carolina X-Ray Society
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

Tennessee Radiological Society
Secretary, Dr. J. M. Frère, 707 Walnut St., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. R. P. O'Bannon, 650 Fifth Ave., Fort Worth 4, Texas. Next meeting, Dallas, Texas, February 3 and 4, 1950.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets first and third Thursdays 4:00 to 5:00 P.M., September to May inclusive. Room 203, Service Memorial Institute, 426 N. Charter St., Madison.

Utah State Radiological Society
Secretary, Dr. Angus K. Wilson, 343 S. Main St., Salt Lake City 1, Utah. Meets third Wednesday in September, November, January, March and May.

Virginia Radiological Society
Secretary, Dr. P. B. Parsons, Norfolk General Hospital, Norfolk, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Homer V. Hartzell, 310 Stimson Bldg., Seattle 1, Wash. Meets fourth Monday each month, October through May, College Club, Seattle.

X-Ray Study Club of San Francisco
Secretary, Dr. W. F. Reynolds, University of California Hospital, San Francisco. Meets from January to July, 1949, at Lane Hall, Stanford University Hospital, and from July to December 1949, at San Francisco Hospital.

Cuba

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

Mexico

Sociedad Mexicana de Radiologia y Fisioterapia
General Secretary, Dr. D. P. Cossio, Marsella No. 11, Mexico, D. F. Meets first Monday of each month.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Ordinary meeting, on the Thursday preceding the third Friday, October to May at 8:15 P.M.
Medical Members' meeting, on third Friday in each month at 5:00 P.M., 32 Welbeck St., London, W 1.

Faculty of Radiologists
Honorary Secretary, Dr. J. F. Bromley, 45, Lincoln's Inn Fields, London, W.C.2, England.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London.

Canadian Association of Radiologists
Honorary Secretary, Dr. E. M. Crawford, 1535 Sherbrooke St., West, Montreal 26, Que. Meetings January and June.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Française d'Electrologie et de Radiologie Médicales
Secretary, Dr. Origéne Dufresne, 4120 Ontario St., East, Montreal, P. Q.

Australian and New Zealand Association of Radiologists
Honorary Secretary, Dr. Alan R. Colwell, 135 Macquarie St., Sydney, N.S.W.
Honorary Secretaries, State Branches:
New South Wales, Dr. E. W. Frecker, 135 Macquarie St., Sydney.
Victoria, Dr. T. I. Tyrer, 3 Lockerbie Court, East St. Kilda.
Queensland, Dr. J. Adam, 131 Wickham Terrace, Brisbane.
South Australia, Dr. R. de G. Burnard, 170 North Terrace, Adelaide.
Western Australia, Dr. A. M. Nelson, 179-B St. Georges Terrace, Perth.
New Zealand, Dr. E. G. Lynch, 12 Bolton St., Wellington.

South America

Sociedad Argentina de Radiologia
Secretary, Dr. Guido Gotta, Buenos Aires, Argentina. Meetings are held monthly.

Sociedad Argentina de Radiología, Filial del Litoral
President, Dr. Francisco P. Cifarelli. Meets second Wednesday each month, at 7:00 P.M., at 663 Italia St. in Rosario.

Sociedade Brasileira de Radiologia Medica
Secretary, Dr. Nicola Caminha, Av. Mem de Sa, Rio de Janeiro, Brazil. Meets monthly, except during January, February and March.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigadeiro Luiz Antonio, 644 São Paulo, Brazil. Meets monthly on second Wednesday at 9 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Peruana de Radiologia
Secretary, Dr. Julio Bedoya Paredes, Apartado, 2306, Lima, Peru. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta, 218, Lima.

Sociedad de Radiologica, Cancerologia y Fisica Medica del Uruguay
Secretary, Dr. Arias Bellini.

Continental Europe

Société Belge de Radiologie
General Secretary, Dr. S. Masy, 111 Avenue des Alliés, Louvain, Belgium. Meets monthly, second Sunday at Maison des Médecins, Brussels.

Ceskoslovenská společnost pro röntgenologii a radiologii v Praze
Secretary, Dr. Roman Bláha, Praha XII, stát. nemocnice, Czechoslovakia. Meets monthly except during July, August, and September. Annual general meeting.

Polish Society of Radiology
Secretary, Dr. L. Zgliczynski, 59 Nowogrodzka St., Warsaw, Poland. Next meeting, Krakow, June 2 and 3, 1949.

Gdansk Section, Polish Society of Radiology
Secretary, Dr. A. Smigielska, Akademia Lekarska, Gdansk. Meets monthly last Sunday at 10.30, X-Ray Dept., Akademia Gdansk.

Warsaw Section, Polish Society of Radiology
Secretary, Dr. L. Zgliczynski, 59 Nowogrodzka St., Warsaw, Poland. Meets monthly.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banual Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad.
USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock, State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly, first Monday, 8 P.M.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

Schweizerische Röntgen-Gesellschaft (Société Suisse de Radiologie)
President, Dr. H. E. Walther, Gloriastr. 14, Zürich, Switzerland.

Societa Italiana di Radiologia Medica
Secretary, Prof. Mario Ponzio, Ospedale Mauriziano. Torino, Italy. Meets biannually.

SCIENTIFIC EXHIBIT

The Scientific Exhibit at the Fiftieth Annual Meeting of the American Roentgen Ray Society held in Cincinnati, Ohio, October 4 to 7, 1949, was among the best that the Society has had presented, and the Scientific Exhibit Committee, under the Chairmanship of Dr. C. Allen Good, gathered together a most representative group of exhibits and the Committee had also cooperated thoroughly by furnishing space accommodations very close to the Commercial Exhibit halls. The various exhibitors themselves contributed in no small way to the success of the exhibits by giving generously of their time in explaining the purport and the details of their exhibits; and the attractiveness of the display and the scientific interest of the various subjects chosen was stimulating and undoubtedly accounted for the popularity of the exhibits this year. The Society proposes to change the viewing boxes in such a manner as not only to better display the exhibits but to lessen the work entailed in mounting and placing the viewing boxes. This in itself will add a great deal to the future Scientific Exhibit section.

There follows a brief description of the exhibits shown and the various awards given.

An exhibit entitled "Cerebral Arteriography" was presented by C. R. Hughes, M.D., R. E. Wiese, M.D., and J. R. Hannan, M.D., Cleveland Clinic Foundation, Cleveland, Ohio. This consisted of representative cases from a review of 150 cerebral arteriograms, showing localization of tumors by vascular displacement, tumor stains associated with various neoplasms, arteriovenous anomalies and aneurysms. The exhibit included charts giving the diagnostic criteria in such studies, the indications for the study as well as the contraindications, and a detailed chart giving the technique of the cerebral arteriography. The exhibit was beautifully displayed and the cases shown were of extreme interest, demonstrating the value of such a diagnostic procedure. Two types of cases were shown, those with primary vascular lesions and a second group illustrating space-taking lesions.

"Cerebral Arteriography; Cerebral Vessal Catheterization" was the title of an exhibit by Karl F. Kesmodal, Jr., M.D., Dan C. Donald, Jr., M.D., and Stacy L. Rollins, Jr., M.D., Jefferson Hospital, Philadelphia, Pennsylvania. The exhibit consisted of (a) roentgenograms showing selective injection of cerebral arteries with barium paste in cadavers; (b) sketches and photographs of dissected gross specimens; (c) description of a method of cerebral arterial catherization and injection of specific arteries with opaque medium in vivo. There were photographs of the equipment used, and roentgenograms of arteries so injected. In the new technique the common carotid is punctured percutaneously and a small radiopaque catheter is passed under roentgenoscopic control into the internal carotid to the cavernous portion.

An exhibit entitled "Tumors of the Lung; a Pathologic and Roentgenologic Study of Surgical Lesions" was presented by J. R. McDonald, M.D., L. B. Woolner, M.D., C. Allen Good, M.D., and A. H. Bulbulian, D.D.S., Mayo Clinic, Rochester, Minnesota. The exhibit presented the pathologic and roentgenologic manifestations of bronchogenic carcinoma, adenoma of the bronchus, pulmonary hamartoma, alveolar cell tumors, metastatic lesions and a miscellaneous group of rare tumors of the lung. The pathologic histology and gross morphology was demonstrated by means of models and photographs of gross and microscopic specimens. Roentgenograms of each type lesion were included. The exhibit was an extensive and beautifully presented one, and received a Certificate of Merit from the Committee on Awards.

Another exhibit on the lung was "The Captive Lung" by J. E. Lofstrom, M.D., D. A. Koch, M.D., W. M. Tuttle, M.D., and P. V. O'Rourke, M.D., Wayne University College of Medicine, Detroit, Michigan. The exhibit consisted of reproductions of various types of captive lung as a result of organizing hemothorax, empyema, pneumothorax and tuberculous effusion. Serial studies were shown, including the early stage of disease, decortication and results. The exhibit was beautifully arranged and detailed descriptions were given of the cases at the time of admission to the hospital and the subsequent films showing the successive changes following operative interference and re-expansion of the lung.

"Coin-Like Pulmonary Lesions" was the title of an exhibit by C. C. Birkelo, M.D., H. J. Klos, M.D., and W. A. Chickering, M.D.,

Herman Kiefer Hospital, Detroit, Michigan. This consisted of an analysis of 98 consecutive cases discovered to have coin-like lesions in a tuberculosis service. The cases represented those in contact with tuberculosis and those found by chance in survey and pre-employment roentgenograms. A description was given of the criteria for classification. An attempt was made to determine the source and ultimate fate of this type of lesion. Representative types were shown. The display was housed in illuminating shadow boxes specially designed for the exhibit and 14 by 17 inch films were used which added enormously to the value of the exhibit. Summary of the 98 consecutive coin-like lesions: proved tuberculosis, 36 cases; undiagnosed or benign, 60 cases; adenocarcinoma, 2 cases.

"Angiocardiographic Studies of the Pulmonary Artery" was the subject chosen for an exhibit by J. E. Miller, M.D., Parkland Hospital, Dallas, Texas. Much confusion exists concerning the pulmonary conus and the pulmonary artery. Angiocardiographic studies demonstrate clearly that the pulmonary conus or outflow tract of the right ventricle is not seen as a part of the left cardiac border in the posteroanterior view. The bulge along the left cardiac border which may be prominent in at least nine different conditions represents the pulmonary artery. Since it may be either the main pulmonary artery or its left branch, this bulge should be called the pulmonary artery segment. Examples were shown of the various abnormalities of the pulmonary artery.

Benjamin Felson, M.D., and Henry Felson, M.D., University of Cincinnati, College of Medicine, Cincinnati, Ohio, had an exhibit on "Localization of Pulmonary Densities from the Posteroanterior Roentgenograms." In this a method for determining the lobar or segmental location of pulmonary lesions from the frontal view of the chest was described and illustrated. It was shown that when a pulmonary density lies adjacent to the heart, the cardiac border is obliterated; when the pulmonary density lies posterior to the heart, the cardiac border remains visible.

An exhibit entitled "Mediastinal Lesions" was presented by Wm. C. MacCarty, Jr., M.D., M. D. Tyson, M.D., and John E. Withee, Hitchcock Clinic, Hanover, New Hampshire. Included in this were cases histories, roentgenograms and photographs (specimens, or operations, or photomicrographs) of lesions producing mediastinal masses. No attempt was made to include all types of lesions occurring in this area. A wide variety of lesions was shown emphasizing the difficulties encountered in making accurate diagnosis of pathological conditions existing in the mediastinum. This exhibit was awarded a Certificate of Merit.

From the New York Hospital, Cornell Medical Center, New York, John A. Evans, M.D., and Ted R. Smalldon, M.D. had prepared an exhibit on "Mediastinal Emphysema." Mediastinal emphysema probably occurs much more frequently than is generally recognized. It is a complication that presents itself in a great variety of diseases and while usually benign, it may occasionally be fatal. The exhibit consisted of charts, diagrams and film transparencies. The mechanisms of production of mediastinal emphysema and the pathways by which air gains access to the mediastinum were illustrated by chart and diagram transparencies. A series of transparencies of roentgen examinations illustrating the roentgen features of typical examples was also presented together with short case histories and suggestions relative to the importance of early roentgen diagnosis. A tabulation was also shown of a large series of cases from the New York Hospital illustrating the great diversity of conditions in which this complication may occur.

An exhibit entitled "The Angiocardiographic Diagnosis of Operability in Lung Cancer" was presented by Charles T. Dotter, M.D., Israel Steinberg, M.D., and Cranston Holman, M.D., The New York Hospital, Cornell Medical Center, New York. The exhibit comprised angiocardiographic studies of the vascular structures in the presence of lung cancer. Certain findings were demonstrated which point strongly to a diagnosis of inoperability. The angiocardiographic signs of inoperability in lung cancer include: (1) partial or complete stenosis of either branch of the pulmonary artery within the pericardium; (2) occlusion of the mediastinal great veins due to tumor; (3) demonstration of mediastinal metastases by the contrast delineation of deformities of adjacent vascular structures; (4) demonstration of pericardial invasion by tumor.

An exhibit entitled "The Roentgenologic Examination of the Colon" was presented by Clyde A. Stevenson, M.D., Robert D. Moreton, M.D., and Everett E. Seedorf, M.D., Scott and White Clinic, Temple, Texas. The exhibit consisted of three parts: (1) equipment and mate-

rials with explanation of exact procedures necessary for complete examination of the colon; (2) nine plastic moulages simulating actual roentgenoscopic examinations and disclosing various pathologic changes which may be encountered; (3) moulages of the pathologic specimens which were demonstrated by roentgen examination, and films illustrating practically all types of neoplastic lesions of the colon. The plastic moulages demonstrated the necessity of oblique projections in order to demonstrate lesions of the colon. This is an excellent teaching exhibit and was beautifully conceived and executed. This exhibit received a Certificate of Merit.

"Diagnosis of Lesions in and about the Fundus of the Stomach" was the title of an exhibit by William H. Smith, M.D., Edsel S. Reed, M.D., and Everett L. Pirkey, M.D., University of Louisville, School of Medicine, Louisville, Kentucky. It consisted of the reproduction of films illustrating the uses of an air-filled balloon as an additional aid in the differential diagnosis of lesions in and about the fundus of the stomach. The following advantages are claimed: (1) definite differentiation between intragastric and extragastric masses under the rib cage; (2) a means of obtaining good double contrast roentgenograms of the stomach; (3) controlled intragastric pressure at any location in the stomach; (4) a means of indirect palpation of the fundus of the stomach is now available. A paper on this subject was published in the July, 1949, issue of the JOURNAL.

An exhibit entitled "Routine Operative Cholangiography" was presented by Maurice D. Sachs, M.D., and P. F. Partington, M.D., Crile Veterans Administration Hospital and Western Reserve University School of Medicine, Cleveland, Ohio. A portion of the exhibit gave the indications for technique and results of operative cholangiography. The remainder of the exhibit consisted of reproductions of representative cases, together with sketches illustrating important points and pertinent operative findings, such as calculi, odditis, pancreatitis, and tumors.

"New Concepts of the Etiology and Surgical Treatment of Hirschsprung's Disease" was the title of an exhibit by Orvar Swenson, M.D., E.B.D. Neuhauser, M.D., and Harold F. Rheinlander, M.D., The Children's Medical Center, Boston, Massachusetts. A series of roentgenograms demonstrated an area of narrowing and dysfunction in the rectosigmoid with dilatation of the colon above this. Recordings of colon peristalsis were demonstrated and drawings of new operative technique and results in the treatment of 30 cases were shown. This exhibit demonstrated in a remarkable manner the new concepts of the etiology and treatment of Hirschsprung's disease. The exhibit was illustrated with roentgenograms of barium enema studies, colored photographs of the specimens and photographs of some of the subjects before and after operation showing the remarkable results obtained from such a surgical procedure. This exhibit was awarded a Certificate of Merit.

An exhibit entitled "Gastrointestinal Roentgenographic Observations in Post Vagotomy" was given by Charles A. Priviteri, M.D., Lawson Veterans Administration Hospital, Chamblee, Georgia. The exhibit was based on work performed on 47 cases of peptic ulcer, 40 being duodenal ulcers and 7 jejunal ulcers. The work was a combined venture between the roentgen-ray and surgical services at the Bronx Veterans Administration Hospital, New York, from July, 1946, to June, 1948. The follow-up and all roentgen examinations on these cases were done by the author. The shortest follow-up was three months and the longest three years, the average of all cases being two years. The studies included preoperative gastrointestinal series on all selected cases and postoperative gastrointestinal series at two weeks, one, three, six, twelve, eighteen, twenty-four and thirty months. Additional roentgenograms were obtained for study of the esophagus, small intestine, colon, gallbladder, and intravenous pyelogram. These examinations were performed at various times after vagotomy in order to obtain a representative response of each organ to vagotomy. One of the important conclusions drawn from their roentgen studies is that vagotomy without gastroenterostomy is no more effective for the treatment of peptic ulcer than any other method.

The exhibit "Alimentary Tract Obstruction in Infancy" by William A. Evans, Jr., M.D., and George A. Miller, M.D., Children's Hospital of Michigan, Detroit, Michigan, was a presentation of roentgenograms showing atresias in the alimentary tract at various levels from the esophagus to the anus, several instances of intrinsic stenosis and obstructions resulting from malrotation and duplications.

Roentgenograms were also shown to illustrate meconium ileus, hypertrophic pyloric stenosis, intussusception, and several types of functional alimentary tract obstruction.

"Cysts of the Kidneys" was the title of an exhibit by Harold C. Ochsner, M.D., Methodist Hospital, Indianapolis, Indiana. There were shown pyelographic findings characteristic of various types of renal cysts found in the patients of a large private general hospital in a five year period.

An exhibit entitled "Porphyria: A Cause of Neuromuscular Small Intestinal Disturbance" was presented by Samuel H. Fisher, M.D., and Robert R. Stanley, M.D., Greenville General Hospital, Greenville, South Carolina. The exhibit consisted of five mounts which discussed the disease and presented a proved case of porphyria. Porphyria is a disease in which an inborn error of pigment metabolism allows the excess production of porphyrins. In porphyria, the porphyrins produce a severe small intestinal disturbance by interfering with the nerve impulses to the small intestine or by irritating the musculature directly. Porphyria as a cause of small intestinal disturbance has apparently not been previously noted in the American roentgenologic literature.

An exhibit entitled "Differential Diagnosis of Abdominal Tumors" was shown by Samuel Brown, N..D., and Archie Fine, M.D., Jewish Hospital, Cincinnati, Ohio. The differential diagnosis of extragastroenteric tumors is based upon certain fundamental anatomical principles, namely: (1) The stomach, intestines and solid organs, within the limits of normal variations, occupy a constant position in the abdominal cavity and maintain an invariable relationship to each other. (2) The stomach and intestines are, relatively speaking, freely movable structures. Their position, shape and the distribution of their contents depend upon the position, shape and size of their neighboring structures and the position of the body as a whole. (3) In the presence of an enlarged organ or an abdominal mass, alterations may take place in the position and shape of the stomach and intestines. These alterations are more or less characteristic, depending upon the organ which is involved. (4) A knowledge of these distinguishing characteristics has enabled the authors to discover the presence of tumors and determine with a high degree of accuracy their exact origin. (5) The roentgen technique which was applied in this study depends upon the three dimensional method which makes use of the anterior, posterior, left and right lateral horizontal positions. (6) A number of roentgenograms were shown to illustrate various abdominal tumors as revealed by this method.

"Polypoid Lesions of the Colon" was the title of an exhibit by Robert M. Potter, M.D., Northwestern University Medical School, Chicago, Illinois. The exhibit demonstrated (1) certain experimental work using phantoms and experimental lumens, showing desirable barium densities and technical factors to best record on roentgen films small intraluminal masses in the colon; (2) certain clinical cases with history, roentgen studies and pathological specimens of polypoid lesions of the colon. There was included in the exhibit a compression device designed by Dr. Hollis Potter which incorporates within a wire frame a bladder which is filled and controlled by a rubber bulb and valve. This compression device has been found very useful in gastroinetstinal examinations, used most often in roentgenographing the patient in the prone position or in oblique angles. Regulated pressure can be used over the distal portion of the stomach and duodenum to study the mucosal pattern. This apparatus has been particularly helpful in separating overlapping groups of redundant sigmoid, colon and in thinning barium by compression over sites of known or suspected bulb lesions and permits better differentiation of the lesion.

An exhibit entitled "Barium Enemas: The Value of Tannic Acid and Post-evacuation Films" was presented by Arthur C. Christie, M.D., Fred O. Coe, M.D., Aubrey O. Hampton M.D., and George M. Wyatt, M.D., The Radiological Clinic of Drs. Groover, Christie and Merritt, Washington, D.C. The authors have used tannic acid in conjunction with post-evacuation films as an integral part of the barium enema examination in over six thousand cases over a period of two years. Comparison of post-evacuation films taken during this period with films taken before institution of the use of tannic acid leaves no doubt that tannic acid produces a more accurate anatomical study of the colon. In substantiation of this finding, the incidence of polyps alone in their clinic has increased over 500 per cent during the two year period. The exhibit consisted of numerous illustrations of the normal and abnormal colon as seen on post-evacuation films

made following the use of tannic acid in the barium mixture. Interpretation of these roentgenograms necessitates a new and different conception of the appearance of the normal and abnormal intestine.

An exhibit entitled "Late Results of Radiation Therapy of Bone Lesions" was shown by T. Leucutia, M.D., Department of Radiology, Harper Hospital, Detroit, Michigan. This exhibit consisted of a series of photographs illustrating the late results obtained by radiation therapy in various types of bone lesions. The material was divided into the following groups: I, sarcomatous tumors including osteogenic sarcoma, chondrosarcoma, Ewing's sarcoma, multiple myeloma and angioendothelioma; II, metastatic tumors from primary malignancies of of different locations such as carcinoma of the breast, carcinoma of the prostate, lymphosarcoma and Hodgkin's disease; III, giant cell tumors; IV, bone cysts, including simple cysts, infective dental cysts, silent cysts and fibrous dysplasia; V, benign tumors including ossifying hematoma, osteoid osteoma, recurrent osteochondroma, multiple chondroma, hemangioma, angiochondroma and myxoma; VI, metabolic disturbances, such as Gaucher's disease, and VII, granulomatous histiocytic lesions such as Schüller-Christian disease and eosinophilic granuloma. In the majority of the cases the final result was shown ten to twenty-five years after the treatment. Photographs of the original roentgenograms in each case were contrasted with those of the roentgenograms illustrating the final results so as to bring out the complete degree of bone restoration which had occurred. Photomicrographs of the histopathologic sections were also presented. This exhibit was awarded a Certificate of Merit.

"Diagnostic Tracer Technique: The Application of Radioactive Tracers to Localization and Differential Diagnosis of Various Lesions" was the title of an exhibit by Lawrence Reynolds, M.D., Kenneth E. Corrigan, Ph.D., Henrietta S. Hayden, Ph.D., and J. O. Reed, M.D., Harper Hospital, Detroit, Michigan. The exhibit illustrated the technical application of a radioactive tracer to some specific diagnostic problems, particularly the differentiation of mediastinal masses and the early detection of cancer of the thyroid and its metastases. The major considerations involving the chemistry, the physiology and the technique of utilization of tracers were shown. One side of the exhibit frame illustrated technical limitations due to the interfering action of medications which include iodine, potassium thiocyanate, sulfa drugs or other chemotherapeutic agents which inhibit the action of the tracer. A second panel illustrated the detection of a malignant lesion and its metastases. The third panel illustrated the differentiation of mediastinal masses by use of the radioisotope. The fourth panel illustrated the problem of the mixed thyroid gland, which includes toxic localizations with non-toxic enlargements, producing a complex diagnostic problem. A model was shown which simulates various types of thyroid lesions and a Geiger counter was available to illustrate the method of detection. The type of probe necessary which was specially developed for diagnostic tracer technique was part of the equipment. Composite charts illustrating typical uptake and excretion in the various types of thyroid disorders were shown. This exhibit was awarded the Bronze Medal.

An exhibit entitled "Scattered Radiation in Fluoroscopic Rooms" was shown by Russell F. Cowing and Charles K. Spalding, New England Deaconess Hospital, Boston, Massachusetts. Measurements have been taken of scattered radiation from the fluoroscope in horizontal and upright positions, using a phantom in place of the patient and with varying size of port. The room size has been varied by use of temporary partitions and the wall material has also been varied. The exhibit was presented by a series of charts and photographs.

An exhibit entitled "Lead Glass Fabric Protective against Roentgen and Beta Rays" was presented by V. W. Archer, M.D., George Cooper, Jr., M.D., H. D. Hebel, M.D., John G. Kroll, M.D., and D. A. Cunningham, M.D., University of Virginia, Charlottesville, Virginia. The high incidence of leukemia among radiologists, and the experimental production of leukemia by irradiation in mice, certainly strongly indicates a connection. Measurements were made on stray radiation received in various parts of the body, and a gown was developed of lead glass fibers to protect the body where not protected by the usual lead rubber apron. The gown covers the arms and drapes down over the legs so that the entire body which by tests receives an appreciable amount of radiation is protected. Protection against beta radiation of atomic fission products was also demonstrated. Two plies of this fabric cuts out 85 per cent of

beta radiation from P^{32}. Each ply weighs 28 ounces per square yard. The Committee on Awards selected this the outstanding exhibit at the meeting and awarded it the Silver Medal.

"Roentgenographic Facsimile" was the title of an exhibit by J. Gershon-Cohen, M.D., and A. G. Cooley, Chester County Hospital, West Chester, Pennsylvania. This included an improved facsimile apparatus for rapid transmission of roentgenographic and cardiographic facsimiles over commercial telephone wires or radio. The exhibit comprised a demonstration of the apparatus in operation.

Cesare Gianturco, M.D., Carle Hospital Clinic, Urbana, Illinois, had an exhibit entitled "Small Cassette Changer". This cassette changer holds six 10 by 12 inch cassettes, measuring $1\frac{3}{4}$ by 24 by 13 inches. A cassette can be exposed every half second or at longer intervals. The position of the patient can be checked roentgenoscopically before exposure.

"The Rapidograph and Its Clinical Applications" was the title of an exhibit by Wendell G. Scott, M.D., Washington University School of Medicine, St. Louis, Missouri. The exhibit consisted of a series of photographs and diagrams illustrating the development and construction of the rapidograph. The techniques that are used with the rapidograph in cardiovascular angiography, aortography, cerebral angiography and in arteriography were described and examples of each were shown together with a beautiful series of films. It has proved of great advantage in preoperative diagnosis both in heart conditions and intracranial conditions because it offers an improvement over previous techniques. The films are made automatically every half second on light, compact, easily handled equipment. Another advantage is the use of a Bucky grid providing greater clarity and definition in the films. The roentgenograms are taken on a roll film which is a recent development, being an adaptation of aerial photographic equipment. This exhibit was awarded a Certificate of Merit.

The Committee on Scientific Exhibits of the American Roentgen Ray Society presented an exhibit entitled "A Comparison of Fluorescent and Incandescent Lighting for Transillumination of Roentgenograms and Colored Transparencies." The Scientific Exhibits Committee had been instructed to investigate the advisability of using fluorescent lighting in standard exhibit frames. Fluorescent lamps, while giving off light of a slightly different character from that emanating from incandescent types, require less current and generate less heat for the same quantity of light. The exhibit consisted of two parts. Colored and black and white transparencies were duplicated in each part, one illuminated by incandescent and one by fluorescent lamps. Members of the Society were asked to compare the two types of illumination and indicate their preference. Also included in the exhibit was a two-sided illuminator using fluorescent lighting which had been constructed for the Radiological Society of North America. The American Roentgen Ray Society is indebted to the Radiological Society for the opportunity to inspect this new type of illuminator. For his part in the designing of this new type of viewing box, Clarence E. Hufford, M.D., Toledo, Ohio, was awarded a Certificate of Merit.

"Bone Metastases Occurring in 600 Cases of Carcinoma of the Uterine Cervix" was the title of an exhibit by Edwin L. Lame, M.D., and Joseph H. Hanson, M.D., Jeanes Hospital, Philadelphia, Pennsylvania. With additional Cases Loaned by Eugene P. Pendergrass, M.D. The discovery in recent years of an unusual number of bone metastases in uterine cervical cancer prompted a review of the 600 cases given treatment for this disease in a twenty year span of one tumor hospital. Comparable analyses are rare in the literature. A study was made of the symptoms, chronology, roentgen characteristics, radiation treatment and prognosis. Skeletal metastases occurred in 0.8 to 7 per cent in other series; 14 out of 600 cases in their own series. Diagrams were presented showing the probable routes through the vertebral veins in accordance with the work of Batson.

An exhibit entitled "Roentgen Study of Soft Tissue Pathology in and about the Knee Joint" was presented by Raymond W. Lewis, M.D., Hospital for Special Surgery, New York, New York. This consisted of a roentgenographic study of normal and abnormal soft tissues in and about the knee joint, with a discussion of the diagnostic features.

"Uterosalpingography" by Tom M. Fullenlove, M.D., Franklin Hospital, San Francisco. California, consisted of a demonstration of cases of sterility showing minor pathological changes and anomalies. Special emphasis was put on the demonstration of the cervical canal and its relation to sterility. This portion of

uterosalpingography is neglected in the usual studies.

"Vitamin D-Resistant Rickets" was the title of an exhibit by John F. Holt, M.D., and Arthur L. Tuuri, M.D., University of Michigan, Ann Arbor, Michigan. A relatively rare form of rickets occurs usually first recognized in the post-infantile period. Although the roentgenologic findings are essentially those of other types of rickets, certain features suggest the diagnosis if it is kept in mind. Differentiation from renal rickets is extremely important because patients with the refractory variety respond remarkably to large doses of vitamin D. Roentgenograms of eight different patients were included in the exhibit.

An exhibit on "Degenerative Disease in Diabetics" was presented by J. H. Marks, M.D., and M. A. Kellett, M.D., New England Deaconess Hospital, Boston, Massachusetts. The exhibit was concerned with the study of a group of 250 patients whose diabetes began before the age of fifteen and who have now been followed fifteen or more years. Attention was directed particularly to the high incidence of arterial calcification among these patients, and to the calcification of the vas deferens and neuropathic joints. Statistical charts also referred to retinal, cardiac and renal damage among the diabetics. The patients were those of Dr. E. P. Joslin and associates.

"Urethane Therapy in Multiple Myeloma" was the title of an exhibit by Wayne Rundles, M.D., and Robert J. Reeves, M.D., Duke University Medical School, Durham, North Carolina. The exhibit consisted of (1) a chart "Pathogenesis of Multiple Myeloma"; (2) bone marrow photographs before and after therapy; (3) charts of hematologic and protein responses to therapy; (4) roentgenograms of skeletons selected from 20 patients, and (5) roentgenograms showing skeletal recalcification after therapy. Charts were also shown giving blood serum and urinary protein changes following urethane therapy.

An exhibit of "Military Field Roentgen-Ray Equipment" was presented by the United States Army and the National Bureau of Standards, Washington, D. C. This exhibit consisted of illustrations of the new light weight roentgen-ray field table and transportable roentgen-ray equipment for general, station and evacuation hospital units. This equipment has taken into consideration units to be used in any and all climates. The equipment includes transportable temperature control roentgen-ray film processing units, and the equipment is designed in such a manner that the units are collapsible and can be transported in a very small space. The units are also so designed that they may be utilized in hospitals of the smallest number of patients as well as hospitals demanding larger requirements.

COMMERCIAL EXHIBIT

The Commercial Exhibit at the Fiftieth Annual Meeting of the American Roentgen Ray Society held in Cincinnati, Ohio, October 4 to 7, 1949, was in point of size larger than any held at a former meeting of the Society. Thirty-eight manufacturers had reserved a total of seventy-seven booths. The display filled the Hall of Mirrors with a number in the adjoining foyer and also filled the South Hall on the floor above. Much new apparatus which had been devised since the last meeting was on display. A great deal of thought and effort had been put on the exhibit of each manufacturer and the combined effect was a truly pleasing one and combined with the practical side made the exhibit a focal point which drew the attention of the members and guests. The exhibit halls were practically filled to capacity during the periods set aside for viewing the exhibits but even at other hours of the day the booths seemed well filled with interested radiologists.

The Society extends its thanks to the commercial exhibitors for their large part in making the Cincinnati meeting a most successful one.

The following firms were represented in the Commercial Exhibit:

Allis-Chalmers Manufacturing Company, Milwaukee, Wis.; *Ansco*, Binghamton, N. Y.; *Automatic Seriograph Corporation*, College Park, Md.; *George W. Borg Corporation, Gibbs Division*, Delavan, Wis.; *Bracke-Seib X-Ray Company*, New York, N. Y.; *Buck X-Ograph Company*, St. Louis, Mo.; *Coreco Research Corporation*, New York, N. Y.; *E. I. du Pont de Nemours and Company*, Wilmington, Del.; *Eastman Kodak Company*, Rochester, N. Y.; *Eldo-*

rado, Mining and Refining, Ottawa, Ontario; *Eureka X-Ray Tube Corporation*, Chicago, Ill.; *Fairchild Camera and Instrument Corporation*, Jamaica, N. Y.; *H. G. Fischer and Company*, Franklin Park, Ill.; *Franklin X-Ray Corporation*, Philadelphia, Pa.; *General Electric X-Ray Corporation*, Milwaukee, Wis.; *Grune and Stratton*, New York, N. Y.; *Paul B. Hoeber*, New York, N. Y.; *Kelley-Koett Manufacturing Company*, Covington, Ky.; *Lea and Febiger*, Philadelphia, Pa.; *Liberty Dressing Company*, Gloversville, N. Y.; *Liebel-Flarsheim Company*, Cincinnati, Ohio; *Machlett Laboratories*, Springdale, Conn.; *F. Mattern Manufacturing Company*, Chicago, Ill.; *Micro X-Ray Recorder*, Chicago, Ill.; *C. V. Mosby Company*, St. Louis, Mo.; *Thomas Nelson and Sons*, New York, N. Y.; *North American Philips Company*, Mt. Vernon, N. Y.; *Pako Corporation*, Minneapolis, Minn.; *Picker X-Ray Corporation*, New York, N. Y.; *Professional Equipment Company*, Chicago, Ill.; *Schering Corporation*, Bloomfield, N. J.; *Frank Scholz X-Ray Engineering Service*, Boston, Mass.; *Standard X-Ray Company*, Chicago, Ill.; *Charles C Thomas, Publisher*, Springfield, Ill.; *Victoreen Instrument Company*, Cleveland, Ohio; *Webster Photocraft*, Webster Groves, Mo.; *Westinghouse Electric Corporation*, Pittsburgh, Pa.; *Winthrop-Stearns*, New York, N. Y.

NEW OFFICERS
AMERICAN ROENTGEN RAY SOCIETY

At the Fiftieth Annual Meeting of the American Roentgen Ray Society held at the Netherland-Plaza Hotel, Cincinnati, Ohio, October 4–7, 1949, the following officers were elected for the year 1949–1950: *President-Elect:* B. P. Widmann, Philadelphia, Pennsylvania; *1st Vice-President:* A. E. Childe, Winnipeg, Manitoba; *2nd Vice-President:* Robert C. Pendergrass, Americus, Georgia; *Secretary:* Barton R. Young, Philadelphia, Pennsylvania; *Treasurer:* Wendell G. Scott, St. Louis, Missouri (re-elected); *Member of the Executive Council:* Earl E. Barth, Chicago, Illinois. The *President* is U. V. Portmann, Cleveland, Ohio, and the *Chairman of the Executive Council* is Milton J. Geyman, Santa Barbara, California.

COURSES IN THE TECHNIQUE OF USING RADIOISOTOPES AS TRACERS

The Special Training Division of the Oak Ridge Institute of Nuclear Studies announces that three additional basic courses in the techniques of using radioisotopes as tracers will be held during the winter of 1950.

The courses will be a continuation of a series offered at Oak Ridge during the past year and a half. The courses will run from January 2–27, January 30 to February 25, and March 6–31, 1950. Thirty-two participants will be accepted for each course. Application for the January 2 course should be mailed prior to December 1, 1949.

Each session is divided into laboratory work, lectures on laboratory experiments, general background lectures, and special topic seminars. Experiments will be conducted covering the use and calibration of instruments and the purification and separation of radioactive materials from inert materials and from other radioactive materials. Other laboratory work will be devoted to the application of various radioisotope techniques. Ample time is allowed for library work and conferences with the staff on individual problems.

Seminar topics include the use of tracers in animal and human experimentation, design of radiochemical laboratories, dosimetry, instrumentation, the effects of radiation on living cells, and the principles and practices of radiation protection.

A registration fee of $25.00 is charged for each participant. Hotel or dormitory facilities are available in Oak Ridge at standard rates and food may be obtained in local restaurants and cafeterias.

Application forms and additional information on the courses may be obtained from Dr. Ralph T. Overman, Chairman, Special Training Division, Oak Ridge Insti-

tute of Nuclear Studies, P. O. Box 117, Oak Ridge, Tennessee.

DR. PENDERGRASS AS VISITING PROFESSOR OF RADIOLOGY

Dr. Eugene P. Pendergrass of the University of Pennsylvania is spending the week of November 14, 1949, in Louisville as the Visiting Professor of Radiology at the University of Louisville School of Medicine under the auspices of the Commonwealth Fund.

EASTERN CONFERENCE OF RADIOLOGISTS

The next meeting of the Eastern Conference of Radiologists will be held in Boston, Massachusetts, on Friday and Saturday, March 3 and 4, 1950, as voted by the delegates of the participating societies at the Washington meeting. The Local Committee is preparing an excellent program. Complete details will appear at an early date.

George Levene, M.D., *Secretary*
New England Roentgen Ray Society

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

Intracranial Tumors. By Percival Bailey, Professor of Neurology and Neurological Surgery, University of Illinois. Second edition. Cloth. Price, $10.50. Pp. 478, with 155 text figures and 16 plates. Springfield, Illinois: Charles C Thomas, 1948.

The monograph by Percival Bailey dealing with intracranial tumors is undoubtedly the most satisfactory one written on this subject. The author has stated clearly and adequately the major points which have to do with the classification, pathology, diagnosis and treatment of these disorders. Moreover, the style of the author is engaging and easy to read, which is in contrast to some medical monographs which are otherwise admirable.

The reviewer for many years has been interested in the diagnostic errors which have resulted from failure to recognize some of the cardinal manifestations of brain tumors. As Bailey says, the most important step in the recognition of such a condition is "to have the possibility of its presence occur to the physician." He emphasizes that the greatest difficulty arises in differentiating primary vascular disease and syphilis of the brain. I should like to add, with regret, that too often patients with frontal lobe tumors are regarded as victims of psychoneurosis for a considerable period of time before the true basis for their complaints is recognized. It is emphasized, however, if the disease is kept in mind and the proper importance attached to severe and persistent headache, repeated small cerebral insults, convulsive seizures, recurrent attacks of vomiting, and failure of vision, with due attention to the general physical, neurological and funduscopic examinations, the diagnosis should be made. In Bailey's opinion, ventriculography is of value from a diagnostic standpoint as expanding lesions of the brain will distort the shape of the ventriculum. He warns, however, that not infrequently the patient's symptoms may be exaggerated and sometimes death results from its use. For these reasons, he believes that this procedure should be employed only when necessary.

The author believes that the classification of intracranial tumors is important because different clinical entities must be segregated for study. It is his opinion, however, that our knowledge is too scanty at present to group them from a pathological standpoint, or according to the tissues from which they arise. His classification into five main divisions is based on "certain familial resemblances of structure and behavior". These groups are as follows: Encephalic tumors, tumors of the covering cells, hypopysial tumors, dysembryomas, and vascular tumors. It is of interest to note that almost one-half (42.6 per cent) are included under the gliomas which are placed under the main division of encephalic tumors. When arranged according to site, he found that of 1108 intracranial tumors, 508 were cerebral, 286 pituitary, 178 cerebellar, 114 cerebellopontine, and 12 brain stem.

There is a selected bibliography of 521 titles arranged alphabetically in the back of the book. Such a type of bibliography serves a useful purpose but many prefer in addition, that all references be placed at the bottom of the page on which the reference has been made.

In general it may be stated that the book is an attractively written, authoritative, comprehensive statement of our present-day knowledge of intracranial tumors from the pen of an experienced surgeon in his field. It is to be recommended to students, practitioners, internists, and those who have a special interest in neurological surgery.

Cyrus C. Sturgis, M.D.

The Skull, Sinuses and Mastoids: A Handbook of Roentgen Diagnosis. By Barton R. Young, M.D., Professor of Radiology, Temple University Medical School. Cloth. Price, $6.50. Pp. 328, with 141 illustrations. Chicago: Year Book Publishers, 1948.

This latest volume of the Handbooks of Roentgen Diagnosis is an outstanding member of this popular and growing family. The author has covered the entire field concisely, succinctly, and in a very readable manner. Basic informa-

tion about the various diseases and disorders of the skull, sinuses and mastoids is summarized adequately and without the inclusion of trivial details or lengthy discussion of debatable theories on etiology or development.

The illustrations are superb, with the text on the opposite page so that the student can easily glance at the illustration while reading the text. The small neat markers are effectively placed on the illustrations and contribute to speedy examination without disturbing the eye.

The author is to be congratulated on the preparation of this splendid book, and the publishers on a superior job of illustration. This book can be unqualifiedly recommended to students and radiologists alike.

Wendell G. Scott, M.D.

Acute Intestinal Obstruction. By Rodney Smith, M.S., F.R.C.S., Assistant Surgeon, St. George's Hospital, London; Consulting Surgeon, Wimbledon Hospital; Hunterian Professor, Royal College of Surgeons. With a chapter on Radiological Diagnosis, by Eric Samuel, M.D., F.R.C.S., F.F.R., D.M.R.E., Late Radiologist, The Middlesex Hospital, London. Foreword by Rupert Vaughan Hudson, F.R.C.S. Cloth. Price, $5.00. Pp. 259, with 102 illustrations. Baltimore: The Williams and Wilkins Co., 1948.

Those interested in intestinal obstruction will want this book, for into its 259 pages is crowded a wealth of information, clearly written, adequately indexed and excellently documented.

After a foreword by Rupert Vaughan Hudson, F.R.C.S. and a brief history of the surgery of acute intestinal obstructions, the subject is dealt with in four sections; namely, pathology, diagnosis, treatment, and individual varieties of acute intestinal obstruction.

The first seven chapters are devoted to the pathology of intestinal obstructions. After a consideration of general pathologic principles obtaining to the problem of intestinal obstruction, the author discusses in detail the pathogenesis of simple obstruction, closed loop obstruction, strangulation, functional obstruction, peritonitis, and protein deficiency. Considerable attention is given to the mechanisms involved in the abnormal physiology that attends intestinal obstruction. Their effects upon the various segments of the bowel involved are reviewed with particular attention to high and low small intestinal obstructions and obstructions of the colon.

The second section of the book deals with diagnosis. Of the latter six chapters, the first concerns the clinical characteristics of the condition and the following five its roentgen manifestations. Eric Samuel wrote this portion of the book and it contains an excellent review of the subject. Of particular interest is his attempt to classifiy small intestinal obstructions into four "grades" depending upon the number of fluid levels seen and the size of the distended loops of intestine. The concept is thought provoking but requires much more work and analysis of large groups of patients. Worth special attention are the comments that concern roentgenography in the obstructions that develop postoperatively.

The third section deals with treatment. It includes six chapters where the problems of chemotherapy, section drainage, protein deficiency, operations and postoperative treatment are discussed.

The final section is approximately one hundred pages long and includes fourteen chapters. In it, one finds brief, yet comprehensive, discussions of the individual varieties of acute intestinal obstruction. Obstructions due to developmental anomalies, strangulated hernia, volvulus, intussusception and various forms of tumors are included. The obstructions of inflammatory origin, mesenteric vascular occlusions, and the postoperative intestinal obstructions are also discussed. Each type of acute intestinal obstruction receives separate treatment and in each instance, the author attempts to review the mechanics of the obstruction, its differential diagnosis and treatment.

Most of the 102 illustrations are good and enhance the value of the text. The bibliography includes 350 references to the literature. The quality of the paper used is quite satisfactory, the printing permits easy reading, and the book is just large enough to fit in an overcoat pocket. We recommend it without reservation.

Philip J. Hodes, M.D.

Vol. 62, No. 5

ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

Department Editor: George M. Wyatt, M.D., 1835 Eye St., N.W., Washington 6, D. C.

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ROENTGEN DIAGNOSIS

HEAD

Wechsler, I. S., and Gross, S. W. Arteriography in cerebral vascular accidents. *J.A.M.A.*, Feb. 19, 1949, *139*, 502–505.

Encouraged by the results from arteriography in cases of subarachnoid hemorrhage due to vascular anomalies, the authors began to use the method in patients with cerebral vascular accidents. The procedure differentiates cerebral thrombosis from hemorrhage so that treatment with anticoagulants or by surgical intervention, respectively, can be instituted with more certain knowledge of the cause of the cerebral apoplexy.

This report is based on 10 cases (6 of intercerebral hemorrhage, 3 of cerebral thrombosis, and 1 of thrombosis of the internal carotid artery). Experience has strengthened the authors' conviction that the procedure should be carried out early if the patient's condition at all warrants it. The arteriographic demonstration of vascular occlusion or displacement of vessels has safely indicated the type of treatment. Thus, arteriography is looked upon as a measure which can in a certain number of cases hasten recovery and prevent permanent damage.—*Henry G. Moehring, M.D.*

NECK AND CHEST

Warren, Shields, and Feldman, J. D. The nature of lateral "aberrant" thyroid tumors. *Surg., Gynec. & Obst.*, Jan., 1949, *88*, 31–44.

An analysis was made in 57 cases clinically diagnosed lateral "aberrant" thyroid tumors. Six other cases were not included because they proved not to be tumors on histopathologic examination.

In 49 of the 57 cases there was an associated thyroid cancer; 15 per cent of these were not detected clinically. In the remaining 8 cases the thyroid gland was not received in the laboratory, either because it was removed elsewhere or because it was regarded as uninvolved by tumor at operation.

The histopathology of the tumor in the thyroid and in the lateral masses of the neck was identical in all but one instance. It was usually papillary in structure, though the neoplastic growth patterns were markedly pleomorphic.

In 40 of the 57 cases the lateral tumors were found to be metastases to cervical lymph nodes. In the remaining 17 cases there was no microscopic evidence that the lateral tumors were either metastatic or derived from embryonal rests.

These neoplasms tend to occur in a younger age group than does cancer in general or than does thyroid disease. The presenting finding is usually only a swelling or nodule in the neck, with an average pretreatment interval of almost four years.

The disease often recurs locally unless completely eradicated by surgery. Follow-up observations must be made over decades rather than years.

The data indicate that most if not all lateral thyroid tumors of the neck are metastases from cancers of the thyroid.—*Mary Frances Vastine, M.D.*

Lester, Charles W. Tuberculosis of the cervical lymph nodes. The present surgical status. *Surg., Gynec. & Obst.*, Dec., 1948, *79*, 719–724.

Certain significant changes have taken place to alter the incidence of this disease since Dowd compiled his series of 700 personal cases in 1916.

1. Tuberculosis in dairy herds has almost been eliminated thus removing bovine tuberculosis as a factor. (However, it is believed that bovine tuberculosis accounted for only about 30 per cent of Dowd's cases.)

2. The widespread removal of tonsils and the replacement of the old guillotine operation by enucleation of the tonsils removed an important portal of entry not only for tuberculosis but also for pyogenic infections which lessen the resistance of the nodes to lymph-borne or hematogenous infection with tuberculosis.

3. The great decrease in tuberculosis generally has vastly diminished the chances for exposure. In 1915 a positive tuberculin test in a child over two years of age was not considered of great significance but in 1948 a positive tuberculin test at any time in childhood has considerable diagnostic weight.

Tuberculosis of the cervical lymph nodes may occur at any age but is predominantly a disease of childhood. It is not frequently associated with active pulmonary tuberculosis and is present in only about 5 per cent of the patients admitted for tuberculosis to the pediatric tuberculosis division at Bellevue Hospital. Among the adult patients the association is even less. Nevertheless every case of lymph node tuberculosis should be subjected to a thorough search

for tuberculous foci elsewhere. While the nasopharynx may be the portal of entry, it is quite probable that the neck nodes draining this area are infected by way of the blood or lymph streams from an infection originating in the lungs which may be healed, merely delitescent, or possibly active.

Treatment. Roentgen therapy has a wide vogue. Unfortunately, it is used in a haphazard fashion without due regard to its limitations, but when indicated it is a valuable adjunctive treatment. It cannot destroy the *Mycobacterium tuberculosis* and is in no sense a sterilizing procedure. Nor can it cause the disappearance of a cold abscess or of caseation necrosis. Its chief effect is to produce fibrosis which is the method by which the body combats the infection. It is, therefore, of value early before caseation necrosis has developed, and late, when cold abscesses drain through chronic sinuses. Its use should be reserved to these indications and it will then be of great value. To continue irradiation of caseous nodes or of cold abscesses submits the patient to useless irradiation.

The treatment of choice is still complete removal in properly selected cases. The ideal time for operation is while the nodes are still relatively discrete.

The judicious use of streptomycin is promising.—*Mary Frances Vastine, M.D.*

JACKSON, CHEVALIER L., BLADY, JOHN V., NORRIS, CHARLES M., and MALONEY, WALTER H. Cancer of the larynx. *J.A.M.A.*, Dec. 11, 1948, *138*, 1080–1083.

The authors evaluate end results in 612 patients with cancer of the larynx treated at the Temple University Hospital from 1930 to 1947. Statistics for five year survival are based upon 216 determinate patients treated from 1930 to 1942, inclusive. By determinate, the authors imply the patients did not die of other cause within five years of treatment and were traced. Surgically treated determinate patients included 102 patients treated by laryngofissure with 81 per cent, five year survival; 31 treated by laryngectomy with 66 per cent, five year survival and 69 treated by irradiation with 51 per cent, five year survival. The authors state that survival statistics on patients treated by roentgen irradiation included both intrinsic and extrinsic lesions without palpable nodes, and leave the impression that patients submitted to roentgen treatment had a poorer original prognosis than those treated surgically. They suggest that three year statistics have some value and that five year statistics are by no means final.

Emphasis is placed on the value of laryngofissure and the value of treatment of recurrences and metastasis with further five year salvage.

BLADES, BRIAN. Mediastinal tumors. *Surg., Gynec. & Obst.*, Jan., 1949, *88*, 131–133.

The discovery of a mediastinal mass by routine roentgenologic examination with or without associated clinical manifestations demands positive diagnosis. Should no available diagnostic procedure provide an infallible method to determine preoperatively the exact nature of a mediastinal tumor, only two therapeutic methods are available: radiation therapy and surgical extirpation. With few exceptions the only mediastinal tumor which will respond to radiation therapy is one of lymphatic origin (lymphosarcoma, Hodgkin's disease and others).

The chief source of confusion in the diagnosis of anterior mediastinal tumors is the differentiation between lymphatic tumors, teratoid tumors, thymomas and bronchiogenic cysts. Posterior mediastinal tumors are chiefly of one common variety. Neoplasms of neurogenic origin are by far the most common. Often, definite clinical signs of disease indicate the presence of malignant degeneration and inoperability.

Bronchiogenic cysts are now known to be fairly common. Pericardial cysts may occur. Thymomas are relatively rare.

Tumors which respond to roentgen therapy are usually incurable. Others may be amenable to surgical extirpation.

The choice between radiation therapy and surgical intervention will depend first upon the tentative diagnosis and, second, upon the danger of exploratory thoracotomy. If roentgenologic and clinical evidence indicate the presence of a lymphoma a test dose of radiation should be given. If improvement does not follow in due course, thoracic exploration should be carried out unless the general condition of the patient precludes the use of any type of surgical intervention.—*Mary Frances Vastine, M.D.*

EHLER, ADRIANA, and ATWELL, SUSANNA. Gastric cyst of the mediastinum. *J. Thoracic Surg.*, Dec., 1948, *17*, 809–820.

Gastric cysts of the mediastinum are rare, only 27 cases having been reported. Only 2 of these cases were found in individuals over

fifteen years of age, while 19 were in infants under one year of age.

Several theories explaining the development of this congenital abnormality are discussed. The cyst usually protrudes into the right hemithorax (in 21 out of 27 cases). All contain some elements of the gastric wall. Roentgenograms are indispensable in diagnosis of this condition. The dense shadow of the gastric cyst is found in the posterior mediastinum and may extend into the right or left hemithorax. The cyst may contain air if there is a communication with the esophagus or trachea. Fluid levels can be demonstrated frequently. Varying degrees of atelectasis and obstructive or compensatory emphysema of the lung are signs auxiliary of the pressure effects of the cysts. Esophagograms are of great value since displacement or distortion of the relatively mobile esophagus can be demonstrated without risk to the patient. Bronchoscopy and esophagoscopy are rarely indicated and only confirm the knowledge gained from the roentgenograms. Aspiration of fluid from the cyst and demonstration of similarity of fluid produced in the stomach make the diagnosis absolute.

The treatment of gastric cysts of the mediastinum is discussed.

One case is presented by the authors.—*Frederic M. Reis, M.D.*

Graham, Evarts. Bronchogenic carcinoma. (Editorial) *Surg., Gynec. & Obst.*, Jan., 1949, *88*, 129–131.

Statistical studies at the St. Louis City Hospital and at the Charity Hospital in New Orleans have shown that, actually, primary carcinoma of the lung is more frequent than that of the stomach. This is primarily a disease of males (about 6 males to 1 female) which occurs during or after middle age. Cough and blood streaked sputum are the two most common symptoms. If the disease arises in a small bronchus at some distance from the main stem bronchus the patient may have no warning symptoms until the condition is too far advanced to be curable.

The author performed the first successful one-stage removal of a lung in a patient who had a squamous cell bronchiogenic carcinoma arising in the main bronchus of the left upper lobe. This was done in 1933. That patient is still living and well. Since this first case the author has performed 296 operations of this sort of which 184 have been for malignant tumors. One of the most distressing features of the problem is the relatively small number of patients who present themselves with a cancer early enough to permit the removal of the lung. In the author's cases only about 25 per cent have had a pneumonectomy.

The operative mortality has steadily declined. In the early years it was 53 per cent but last year (1947) of 20 pneumonectomies for cancer the author had only 2 deaths, a hospital mortality of only 5.1 per cent.

Summary

1. Bronchogenic carcinoma is one of the most frequent forms of cancer.

2. The operation of total pneumonectomy offers hope to those victims of this disease who, prior to 1933, had no hope.

3. Nowadays the principal problem in improving the results is to have patients come for help early enough to permit the operation.

4. This means education of the profession to the recognition of the frequency of this condition, the necessity of early examinations of a proper kind, and the fact that the early case is curable.—*Mary Frances Vastine, M.D.*

Edwards, Lydia B., Lewis, Ira, and Palmer, Carroll. Studies of pulmonary findings and antigen sensitivity among student nurses. *Pub. Health Rep.*, Dec. 3, 1948, *63*, 1569–1600.

This report is concerned with surveys conducted on student nurses in 76 nursing schools in 10 different, widely separated metropolitan areas. At the time of making the report, more than 23,000 nurses were under study; the object of the investigation is to determine the correlation between pulmonary infiltrates and sensitivity to tuberculin and histoplasmin. Of 12,803 nurses included in the study reported, only 224 showed infiltrates and these were investigated as to sensitivity with regard to type of pulmonary lesion.

Of the 224 showing pulmonary infiltrates, 10 per cent failed to show positive reaction to either tuberculin or histoplasmin; 60 per cent reacted positively to histoplasmin, not to tuberculin; 19 per cent reacted positively to tuberculin, not to histoplasmin and 10 per cent reacted positively to both.

Types of infiltrate were arbitrarily designated as (a) "nodular," which were lesions having more or less circular outline and sharply, clear-cut borders; (b) "poorly circumscribed,"

having poorly defined borders, less dense composition and less homogeneous than the nodular lesions, with borders merging gradually into surrounding lung tissue. Some areas may be fibrotic but some are also "soft"; (c) "fibrosis alone," pulmonary "scar," single or multiple strands, sharply outlined, with no "soft" elements; (d) "non-specific"—gross pneumonias, bronchiectasis, etc. In the group of "nodular" infiltrates, they found 42 per cent of the 224 individuals reacted to histoplasmin, not to tuberculin. In the "poorly circumscribed" group, 10.2 per cent reacted to tuberculin, not to histoplasmin and 12.5 per cent in the "fibrosis alone" group reacted to histoplasmin. While there were some reactors to the other antigen in each group, and to both, the proportion was much smaller. The variability was due to the fact that tuberculin reactors were among those whose infiltrates tended to be in the upper part of the chest with infiltration of less clearly defined nature. The histoplasmin reactors were among those whose infiltrates were scattered generally throughout the lung fields. They noted, also, that there was a tendency for infiltrates associated with histoplasmin sensitivity to be smaller than those with tuberculin sensitivity.

There were 38 who showed mediastinal involvement. Of these only 35 showed calcification; 31 of these reacted positively to histoplasmin, 4 to both histoplasmin and tuberculin, none to tuberculin alone. Of these 35, only 20 showed parenchymal infiltration, and this was most frequently of the nodular type. Three individuals with mediastinal involvement reacted to neither histoplasmin nor tuberculin, none had calcification or parenchymal infiltration.

They were able to follow the course during training of a significant number and found that those with fibrotic type of infiltration showed no change during training. Only one of the group with nodular infiltration showed alteration—this was a transitory increase in size which returned spontaneously to former size within a month. A group of 23 in the "poorly circumscribed" classification reacted to tuberculin alone. Of these, 7 showed increase in size of existing lesions or development of new lesions, were hospitalized or had to discontinue training. Twelve, who reacted to histoplasmin only showed either resolution of lesions, development of more sharply defined margins or conversion to nodular type.

It would appear, therefore, that the majority of nodular lesions are due to histoplasmin infection, the majority of poorly defined lesions, in upper lung fields, are due to tuberculous infection; that the poorly defined group, reacting to tuberculin, are likely to break down and show evidence of activity. The majority of mediastinal node involvements are likely to be due to histoplasmosis infection rather than to tuberculosis.—*Angus K. Wilson, M.D.*

ADAMS, JOHN M. Primary pneumonitis in infancy. *J.A.M.A.*, Dec. 18, 1948, *138*, 1142–1144.

The epidemic occurrence of primary pneumonitis in infants has already been recognized. The authors wish, however, to emphasize the sporadic occurrence of the disease; further, the severity of the disease appears to be dependent upon the age and maturity of the patient. The morbidity and mortality were extremely high in prematurely born infants as compared with normal full term babies. The same striking difference in morbidity is noted between newborn, full term babies and older children and adults.

The symptom pattern of cough, dyspnea, and cyanosis usually manifests itself in the more acutely ill patients. The onset is usually abrupt with sneezing and coughing. The febrile response is usually slight, but it may eventually rise to 103°F. Physical findings are few in the milder cases, but in the more acutely ill, cyanosis and dyspnea may be quite marked with an abundant whitish, thick exudate in the pharynx. Rales may be heard in both lungs.

The diagnosis is made by the symptom pattern and by the rather characteristic shadows seen in the roentgenograms of the lungs. The roentgenogram reveals diffuse shadows which are bronchial in distribution and usually widespread but may be confined to a single lobe of the lung. Pharyngeal smears made in the acute stage of the disease reveal a great predominance of epithelial cells containing cytoplasmic inclusion bodies. This finding is considered as helpful in the diagnosis. The treatment is largely symptomatic. Continuous oxygen is beneficial where there is involvement of the lower respiratory tract. Sulfonamides and antiobiotics are used primarily to combat secondary bacterial complications.

It is suggested that a full term, newborn infant may be more protected from the disease by passive transfer of protective substance from its mother. Clinical experience suggests

that concentrated gamma globulin may contain protective substances against the agent responsible for primary pneumonitis in infants. —*Wallace M. Roy, M.D*

CARSON, M. J., and GOODFRIEND, J. Constricting vascular rings; report of two cases with recurrent respiratory infections. *J. Pediat.*, Feb., 1949, *34*, 155–165.

Double aortic arch in infancy results in respiratory stridor, recurrent respiratory infections, dysphagia and vomiting. The authors report 2 cases in which recurrent respiratory infections were the predominant manifestation. Illustrative roentgenograms are reproduced showing the typical bilateral indentation of the esophagus in the posteroanterior projection and the posterior indentation in the lateral projection.

Tracheograms showed constriction of the trachea above the carina. Operative correction in one case resulted in cessation of the respiratory infections. The parents of the second child refused operation.

The authors postulate that anomalous vascular rings predispose to respiratory infections by narrowing of the trachea and interference with adequate drainage of the tracheobronchial tree. They advise contrast studies of the trachea and esophagus in all patients with recurring pulmonary infections to exclude vascular ring anomalies.—*Rolfe M. Harvey, M.D.*

BEADENKOPF, WILLIAM G., LOOSLI, CLAYTON G., LACK, HERBERT, RICE, FREDERICK A., and SLATTERY, ROBERT V. Tuberculin, coccidioidin and histoplasmin sensitivity in relation to pulmonary calcifications. *Pub. Health Rep.*, Jan. 7, 1949, *64*, 17–32.

The authors report on 6,000 University of Chicago students who were tested routinely with tuberculin, histoplasmin and coccidioidin and the findings correlated with pulmonary calcifications as seen in 70 mm. chest survey films. Films were interpreted without knowledge of antigen sensitivity. Of the tuberculin and histoplasmin 1.4 per cent, and 0.6 per cent of the coccidioidin reactions were regarded as doubtful and not considered in final evaluation; 10 per cent of chest calcifications were regarded as doubtful but, in accordance with practice of other authors, were included as positive in final analysis.

Of the students examined, only 800 were found to have pulmonary calcifications. Of these, 33.6 per cent failed to react to any of the antigens; 25.6 per cent reacted to histoplasmin; 20.3 per cent reacted to tuberculin and 0.9 per cent reacted to coccidioidin. The groups were further studied for combined reactions to the three antigens, and it was found that 11.6 per cent reacted to tuberculin and histoplasmin, 4.8 per cent to histoplasmin and coccidioidin, 2.6 per cent to tuberculin, histoplasmin and coccidioidin and 0.6 per cent to tuberculin and coccidioidin.

In all age groups under fifty years, histoplasmin reactors were found more frequently than tuberculin, in association with pulmonary calcifications. In the group over fifty years of age, there were no purely histoplasmin reactors but those with histoplasmin and tuberculin sensitivity were two and a half times more frequent than those who reacted to tuberculin alone.

From the roentgenological point of view, it would appear that in a given film showing pulmonary calcification, likelihood of etiology would be, in the order named: (*a*) either tuberculosis, histoplasmosis or coccidiomycotic infection, (*b*) histoplasmin infection, (*c*) tuberculous infection, (*d*) various combinations of those three, (*e*) coccidiomycosis. Histoplasmosis is a more frequent cause of pulmonary calcification than is tuberculosis.

They noted that, geographically, both histoplasmosis sensitivity and pulmonary calcifications were more prevalent in the lower Mississippi Basin of the United States.—*Angus K. Wilson, M.D.*

WOOLNER, LEWIS B., and MCDONALD, JOHN R. Diagnosis of carcinoma of the lung; value of cytologic study of sputum and bronchial secretions. *J.A.M.A.*, Feb. 19, 1949, *139*, 497–502.

The sputum or bronchial secretions of 2,188 patients were examined for carcinoma cells. On the basis of the smears, carcinoma was diagnosed in 200 cases. Of these 200 cases: 190 had a final diagnosis of bronchogenic carcinoma or pulmonary metastatic carcinoma; 4 had carcinoma of the esophagus, larynx, or trachea; 4 were false positives; and in 2 the final diagnosis remained uncertain. In 29 of 74 cases of bronchogenic carcinoma surgically explored, the smears supplied the only preoperative microscopic evidence of carcinoma. In 6 cases of Pancoast's tumor, carcinoma cells were found in the sputum.

Of the 4 false positives: 1 had a chronic tuber-

culous process; 1 had pronounced bronchiectasis; 1 chronic pneumonitis; and 1 had extensive infarction plus an organizing pneumonic process.

The accuracy of this technique varies directly with the experience of the cytologist.

Cytologic examination of the sputum is of great diagnostic value in patients suspected of having bronchogenic carcinoma and in whom bronchoscopic examination is contraindicated. —*Henry G. Moehring, M.D.*

SACHS, MILTON E. Acute coronary thrombosis occurring in a case of perforating lesser curvature ulcer, undergoing malignant changes. *Am. J. Digest. Dis.*, Nov., 1948, *15*, 375–378.

Acute coronary occlusion has many times been mistaken for an abdominal catastrophe and, conversely, symptoms due to abdominal conditions may simulate those of a cardiac accident. Diagnosis may be even more difficult if in a case of penetrating ulcer with active symptoms, coronary thrombosis supervenes. In such an instance, one might erroneously attribute the entire symptom complex to an acute perforation and overlook the coronary thrombosis. The following points of interest may be repeated in summing up the case presented: A gastric ulcer may persist for some time without demonstrable roentgen evidence. When coronary thrombosis occurs in a case of a chronic penetrating lesser curvature ulcer during the stage of an acute exacerbation, diagnosis may be difficult. Electrocardiographic studies should be included along with gastrointestinal studies. In the presence of an increased size of the niche the diagnosis of malignancy must be considered and surgery instituted. The occurrence of coronary occlusion with myocardial changes appears to be no contraindication to major surgery, in the absence of cardiac failure.—*Franz J. Lust, M.D.*

DOTTER, C. T., AND STEINBERG, I. The diagnosis of congenital aneurysm of the pulmonary artery. *New England J. Med.*, Jan. 13, 1949, *240*, 51–54.

A local aneurysm or a diffuse aneurysmal dilatation of the pulmonary artery is presumed by the authors to be of congenital origin if the known causes of acquired pulmonary artery enlargement can be ruled out. Causes of acquired enlargement include infections of the artery, mitral stenosis, cardiac failure, and a group of chronic pulmonary disorders accompanied by pulmonary hypertension. Pure congenital pulmonary aneurysm is rare, and should be distinguished from pulmonary artery dilatation secondary to intracardiac septal defect or distal to a pulmonic stenosis.

Two adult patients are described, each with an asymptomatic pulsating mass in the left hilum. Angiocardiograms with neo-iopax showed the mass to be an aneurysmal dilatation of the main stem pulmonary artery, and of one or both of its main branches. The dilated vessel is best demonstrated in the left lateral projection, taken three seconds after beginning the opaque injection.—*Henry P. Brean, M.D.*

NEUHOF, HAROLD, and NABATOFF, ROBERT A. An angiographic study of the form and function of the remaining lung after pneumonectomy. *J. Thoracic Surg.*, Dec., 1948, *17*, 799–808.

This paper begins with a survey of the experimental and clinical studies on the subject. The authors thought that a study of the topography of the pulmonary vessels would throw additional light on the question of the change in form and perhaps offer indirect evidence as to changes in function of the remaining lung. Twelve patients ranging in age from eighteen to sixty-two years, who had undergone pneumonectomy for malignant tumors were utilized for angiographic studies. The intervals after pneumonectomy varied from six months to ten years.

The vascular pattern presented an essentially normal arrangement. There were some minor abnormalities, as follows: (1) Varying degrees of displacement of the pulmonary artery towards the operated side, accompanying the mediastinum. (2) Some angulation and a turning on itself of the uppermost medial branch of the remaining pulmonary artery. (3) Visibility of the vessels at a lower level than usual in the lower lobe, delineating a downward displacement of the diaphragm. This is often the only direction in which the lung can expand, for the dead space on the operated side may become rapidly reduced by the presence of fluid and the upward shift of the diaphragm.

The remaining lung expands, but this represents nothing more than an unfolding of the areas in the lower lobes which normally function only on violent exertion. The vascular pattern in the expanded lung points to simple expansion of the lung, but emphysema (de-

crease of function) was not found in this series of cases.

No alteration in size or recognizable thrombosis occurred in the stump of the resected pulmonary artery.—*Frederick Reis, M.D.*

Burford, T. H., and Carson, M. J. Visualization of the aorta and its branches by retroarterial diodrast injection. *J. Pediat.*, Dec., 1948, *33*, 675–687.

Visualization of the left side of the heart and aorta following intravenous injection of diodrast is usually not so satisfactory as the visualization of the right side of the heart. Various procedures of injecting diodrast directly into the aorta have been devised. The authors experimented with those methods which employ a vessel originating in the aortic arch. The authors consider the left common carotid artery to be the most suitable vessel for the injection in children.

Their technique includes general ether anesthesia for the patient. Under sterile precautions the left common carotid artery is surgically exposed and isolated with three ligatures. The patient is placed on the tautograph in the right posterior oblique position. The upper ligature is tied to prevent cephalad reflux of the dye. The lowermost ligature is tied loosely and an incision made through which a No. 10 cannula is introduced. The middle ligature is tied around the vessel over the cannula to prevent blood leakage. The lowermost tie is loosened and 30–50 cc. of 70 per cent diodrast in injected rapidly.

With this technique the authors have been able to visualize patent ductus arteriosus and coarctation of the aorta. Illustrative films are reproduced.

The authors feel the method is of value in localizing the point of constriction in cases of coarctation and also in demonstrating the feasibility of surgical resection and anastomosis. Congenital anomalies of the arch are easily demonstrable. The method should be useful in the problem of aortic aneurysms.—*Rolfe M. Harvey, M.D.*

Gross, Robert E. Surgical treatment for coarctation of the aorta. *J.A.M.A.*, Jan. 29, 1949, *139*, 285–292.

Gross reports the results of the surgical treatment of coarctation of the aorta in 60 cases.

The most characteristic physical signs of this congenital anomaly are observable in the peripheral circulation. Pulsation in the femoral arteries is greatly diminished or absent. The blood pressure in the lower extremities is less than in the upper. Evidence of collateral circulation is often observed in adults but not in children.

The roentgen signs in infancy and childhood show cardiac enlargement, or other changes which do not indicate the type of cardiovascular abnormality.

Beyond the first decade the roentgen signs are usually pathognomonic. Chiefly the notching or scalloping of the under margins of the posterior and lateral portion of the ribs due to erosion by the pulsating, tortuous intercostal arteries. The upper and lower three ribs are usually not involved.

The aortic knob is small and lacks its usual prominence. Angiocardiography is most useful. The author prefers to introduce the injection of contrast substance directly into the aorta by way of a catheter threaded through the axillary and subclavian arteries.

The surgical techniques are described briefly. Of the cases reported there was complete relief of hypertension in 41. There were 7 deaths from various causes.—*I. Schwartz, M.D.*

Adams, Herbert D., Rutledge, David I., and Souders, Carlton R. Coarctation of the aorta. *J.A.M.A.*, Feb. 5, 1949, *139*, 362–365.

In the past, prognosis for the patients with coarctation of the aorta has been poor because of the propensity for coarctation to result in heart failure, rupture of the aorta, cerebrovascular accident, or subacute bacterial endocarditis. The recent development of a surgical technique for correcting the anomaly must alert physicians to the possibility of coarctation in young persons with hypertension.

Diagnostic points include: (1) absent or diminished pulsations in the abdominal aorta and femoral arteries (collateral circulation may be adequate to produce pulsation in dorsalis pedis and posterior tibial arteries); (2) unusual pulsations of intercostal arteries; (3) when the patient stoops forward with arms hanging by his side, previously invisible collateral arteries may be seen under the skin of the back and sides of the thorax; (4) the arterial pressure in the legs is too low to be measured or, at least, is lower than in the arms, the reverse of normal (hence pediatricians especially are urged to make a comparison of blood pressure in upper and lower extremities during routine general exam-

inations of their patients); (5) a *loud*, rough, systolic murmur may be heard along the left sternal border and, as well, along the left margin of the vertebral column; (6) characteristic notching of lower rib margins on the roentgenogram (in 75 per cent or a series of 43 cases). Angiography, while seldom required for diagnosis, may yield information helpful in planning surgical treatment.

Surgical treatment is safer, and the results are better, when applied during childhood because then the vessels are more elastic and less friable. Gross has placed the age limits from six to twenty-five years for resection of the constricted area and end-to-end anastomosis. In older patients anastomosis of the left subclavian artery to the descending aorta below the coarctation warrants consideration.

The surgical management of 5 cases of coarctation is described.—*Henry G. Moehring, M.D.*

ABDOMEN

GRIMSON, K. S., RUNDLES, R. W., BAYLIN, G. J., TAYLOR, H. M., and LINDBERG, E. J. Vagotomy. *J.A.M.A.*, Feb. 19, 1949, *139*, 508–513.

The experiences, analyzed in considerable detail, are derived from 104 patients who had intractable, frequently recurring, or complicated peptic ulcer and were treated by vagotomy. The conclusions drawn are: (1) Vagotomy without gastroenterostomy has not been a satisfactory surgical treatment for duodenal ulcer. (2) Vagotomy with gastroenterostomy has yielded encouraging results as a treatment for duodenal ulcer. (3) Vagotomy has effected healing of stomal ulcer. (4) Vagotomy has aided in the treatment of benign gastric ulcer, but persistence or recurrence has been frequent. (5) In general, side effects of vagotomy seem to diminish with the passage of time.—*Henry G. Moehring, M.D.*

PAULSON, MOSES, and GLADSDEN, EUGENE S. Gastroscopic appearances following vagotomy. *J.A.M.A.*, Jan. 15, 1949, *139*, 151–152.

In a group of cases gastroscopic examination was done following (1) simple vagotomy, (2) vagotomy with gastroenterostomy and (3) vagotomy with subtotal gastrectomy. In the first group, simple vagotomy, gastric dilatation and atony were the findings with peristaltic activity decreased and with little mucosal change. The authors were surprised to find the pylorus was patulous and continually open rather than spastic as had been surmised before. After a period of a year a tendency toward restoration of normal tone and peristalsis was noted. In the second group, namely vagotomy with gastroenterostomy, the same changes were noted including the gaping of the pylorus. In addition the opening of the stoma tended to be more patulous and less active than seen in gastroenterostomy without vagotomy. Mucosa about the stoma appeared to be somewhat erythematous. In the third group, namely vagotomy and subtotal gastric resection, they found considerable mucosal change in the remaining pouch with erythema and thickening and occasional edema. Again the stomal openings were larger than one usually sees in subtotal gastrectomy and the jejunum showed relatively mild mucosal changes on the one occasion in which it was seen. These observations were controlled using patients without vagotomy and with all other factors the same including preparatory sedation and atropine and no such findings were obtained.—*Arthur J. Present, M.D.*

COLP, RALPH. Surgical treatment of gastric, duodenal and gastrojejunal ulcer, including the present status of vagotomy. *Bull. New York Acad. Med.*, Dec., 1948, *24*, 755–771.

Vagotomy as a sole procedure has been abandoned in the treatment of unobstructed duodenal ulcer as a result of Colp's experiences in a series of 21 cases, because 7 of them required further surgery, 2 for recurrent duodenal ulcer, and 5 for gastric dilatation and atony. The completeness of the division of the vagi, as evidenced by the insulin test, bears no relationship to the clinical results. The addition of gastroenterostomy to vagotomy seems to have eliminated the undesirable effects of gastric atony in 26 cases of duodenal ulcer in which it was performed. Whether the incidence of gastrojejunal ulceration will be lessened by the combination of vagotomy and gastroenterostomy, as compared to gastroenterostomy alone, only long range follow-up studies will determine. Gastroenterostomy combined with bilateral infradiaphragmatic vagotomy is the preferred procedure in cases of duodenal ulcer unsuitable for subtotal gastrectomy.

For Colp the subtotal gastroectomy still remains the operation of choice in duodenal ulcer. It has been combined with infradiaphragmatic vagotomy in a series of patients whose pre-

operative acidity was high, and who had a tendency to bleed. There has been no increase in the operative mortality and a slight increase in the postoperative morbidity attributable to the added vagotomy. Whether the incidence of recurrent gastrojejunal ulceration will be diminished remains a subject for further study.

The immediate results of vagotomy in the treatment of gastrojejunal ulceration following gastroenterostomy and subtotal gastrectomy have been excellent. Subsequent follow-up has revealed recurrent ulceration in some cases. In patients considered to be good operative risks, a subtotal gastrectomy with infradiaphragmatic vagotomy is preferable to vagotomy alone for gastrojejunal ulceration following gastroenterostomy. In gastrojejunal ulceration following subtotal gastrectomy, wherever possible resection of the ulcer and further gastric resection combined with infradiaphragmatic vagotomy would seem preferable to the severance of the vagus nerves alone.

Vagotomy is not indicated in the treatment of gastric ulcer.—*Franz J. Lust, M.D.*

Ricketts, Wm. E., Palmer, Walter Lincoln, Kirsner, Jos. B., and Hamann, Anna. Achlorhydria and peptic ulcer; further study of the role of peptic activity in the pathogenesis and course of peptic ulcer. *Ann. Int. Med.*, Jan., 1949, *30*, 24–39.

Previous studies have tended to establish the invariable presence of acid gastric juice in patients with chronic peptic ulcer and the absence of ulcer in patients with persistent achlorhydria as in pernicious anemia. Murphy and Howard noted roentgenologic evidence of duodenal ulcer in 4 of 440 patients with pernicious anemia; apparently the lesion consisted of roentgenologic deformity (scar) rather than an ulcer crater.

In active duodenal ulcer. the question of achlorhydria or even of a low secretory rate almost never arises. Thus, in each of 500 consecutive patients with active duodenal ulcer reviewed, the maximum response to histamine stimulation was above 40 clinical units. Very occasional instances of duodenal deformity due to scarring have been noted in patients with pernicious anemia or gastric cancer but an active duodenal ulcer under such circumstances has never been seen.

The incidence of prolonged achlorhydria following roentgen irradiation is quite low, just how low is impossible to state at present because of the varying techniques used and the incompleteness of the data. However, adequate information on 139 patients in whom achlorhydria developed is available; complete healing of the ulcer occurred in 134 of these. In the 44 patients with achlorhydria lasting longer than ninety days, complete healing occurred in all. Of the 30 patients with gastric ulcer in whom prolonged achlorhydria was produced, complete healing occurred in all but 2; in these 2 the achlorhydria lasted only two months and two and one-half months respectively. In 102 patients with duodenal ulcer, complete healing occurred in all but 3; in these the achlorhydria lasted less than one month. In 2 cases the initial depression of gastric secretion was not as great as that which occurred later—apparently a delayed reaction due to atrophy of the mucosa resulting from radiation injury. Healing of the ulcer in these 2 cases was somewhat slower but it occurred in both when complete achlorhydria developed.

The conclusions of the authors are: that chronic peptic ulcer occurs only in the association with acid gastric secretion; achlorhydria lasting longer than three months produces complete healing of peptic ulcer irrespective of the age of the patient or the duration of the disease; spontaneous or induced achlorhydria, if permanent, produces permanent healing of peptic ulcer.—*Eugene J. McDonald, M.D.*

Cove, Arthur M., and Curphey, Wilfred C. Prolapse of redundant gastric mucosa. *Surg., Gynec. & Obst.*, Jan., 1949, *88*, 108–114.

In the past the presence of hypermobile redundant gastric mucosa has been such a common and incidental finding by surgeons and roentgenologists that little significance was placed upon it as an etiological factor. Consequently, no attempt was made to correct the condition. Since Scott in a recent article gave impetus to the study of this problem, more and more writers are beginning to recognize its significance as a clinical entity and as the cause of any otherwise unexplained gastrointestinal complaints. It is difficult to understand why this condition should have been so universally overlooked in the past in patients with gastrointestinal disease.

Conclusions

1. Prolapse of gastric mucosa is more common than generally suspected.

2. The diagnosis can only be established

roentgenologically since the clinical picture is extremely variable.

3. Mild degrees of prolapse rarely produce symptoms and probably are only an incidental finding.

4. Moderate or marked degrees of prolapse are often associated with complaints of sufficient severity to cause the patient some degree of disability.

5. The most common complication of prolapse is gastrointestinal hemorrhage.

6. The symptoms of prolapse can be controlled medically in the majority of instances.

7. With symptoms of sufficient degree, failure of response to medical treatment is an indication for surgical treatment. Surgery is also indicated in those patients who have had repeated severe gastrointestinal hemorrhage.

8. The surgical procedure of choice is the simplest which will correct the lesion—antrotomy with removal of redundant gastric mucosa combined with some type of pyloroplasty. —*Mary Frances Vastine, M.D.*

Pollard, H. Marvin, Bryant Henry C., Block, Malcolm, and Hall, Winston C. Diagnosis of gastric neoplasms. *J.A.M.A.*, Jan. 8, 1949, *139*, 71–74.

The authors report their experiences in the diagnosis of gastric neoplasms by the examination of cells from the gastric juice. The study was made on the gastric content of 278 patients complaining of gastrointestinal distress. As nearly as possible, the staining technique advised by Papanicolaou was used throughout. The pathologist who examined all slides found that while some positive slides can be evaluated quickly, twenty minutes or more are necessary before a negative preparation can be discarded. Often, more specific cellular changes make an absolute diagnosis by this method impossible. It is emphasized that there is no single criteria of malignancy in the individual cell which may not occur in the normal cell.

In this study, the diagnosis as made by microscopic study of the gastric juice is compared with the clinical diagnosis. The series includes only those cases in which it was felt that a final diagnosis had been established. In the first group consisting of 192 aspirations there was no clinical evidence of carcinoma of the stomach. In 86 per cent of the aspirations, no malignant cells could be found. In 10 per cent the results were indefinite, while 4 per cent were falsely reported as positive for carcinoma. The second group consisted of 43 patients who were subjected to laparotomy and no histopathological evidence of carcinoma could be found. Normal cells were found in the smears in 72 per cent. Nine per cent of the aspirations were indefinite, while in 19 per cent it was felt that malignant cells were present. In group III 20 aspirations were done on patients in which the final clinical diagnosis was carcinoma but laparotomy was not done and histopathological confirmation was therefore unobtainable. No malignant cells, however, could be found in 60 per cent of these cases. While a positive report was made in only 40 per cent. In the final group of 41 patients, who had histopathological evidence of carcinoma, only 35 per cent of the smears were positive, while 60 per cent were indefinite or negative for carcinoma.

The authors believe that as pathologists gain more experience in the study of the gastric smears, the results may be improved. On a number of occasions, however, where roentgenograms of the stomach have been suspicious, but not conclusive of carcinoma, microscopic studies of the gastric smears revealed definite malignant cells. The diagnosis was substantiated by gastroscopy and proved by laparotomy. In the present state of development, the authors indicate that it is no more than an adjunct to the present accepted methods of examination. —*Wallace M. Roy, M.D.*

McClenahan, J. E., and Fisher, Bernard. Idiopathic megaduodenum. *Am. J. Digest. Dis.*, Dec., 1948, *15*, 414–416.

A case of idiopathic megaduodenum is described. Of approximately 250,000 admissions and 3,500 autopsies in the last twenty five years at the Mercy Hospital, Pittsburgh, this is the only case of proved megaduodenum where no etiologic factors could be found.—*Franz J. Lust, M.D.*

Faxon, H. H., and Schoch, W. G., Jr. Gastrojejunocolic fistula. *New England J. Med.*, Jan. 20, 1949, *240*, 81–87.

Although gastrocolic fistula is sometimes caused by direct extension of carcinoma of the colon or stomach into the adjacent organ, the great majority of gastrojejunocolic fistulas result from perforation into the colon of a gastrojejunal ulcer in a patient who has had a gastroenterostomy for peptic ulcer. The incidence of gastrojejunal ulcers after gastroenterostomy varies considerably in different series, and per-

haps averages 3 per cent. Between 9 and 24 per cent of all marginal ulcers perforate into the colon with resultant gastrojejunocolic fistula. Since subtotal gastrectomy is generally replacing simple gastroenterostomy as a surgical treatment of peptic ulcer, the incidence of marginal ulcers, and of gastrojejunocolic fistulas, is steadily decreasing.

The fistulas vary in diameter from a few millimeters to several centimeters, and are lined with a well formed mucosa. The colon may be locally constricted and partially obstructed. Unless the fistula is large, the disposition of the jejunal mucosal folds prevents passage of gastric contents into the colon, but permits regurgitation of colonic contents into the stomach. This is probably the explanation for the fact that the fistula is much more readily demonstrated by barium enema than by barium given by mouth.

The authors present 9 patients with gastrojejunocolic fistula, averaging forty-five years of age at onset of fistula symptoms. The average symptom-free period following gastroenterostomy was nine years, although some patients had no symptom-free period. The period between gastroenterostomy and development of fistula symptoms varied from one to thirty years, averaging eleven years.

Clinical features, in order of frequency, were weight loss, diarrhea, weakness, nausea or vomiting, anorexia, hemetemesis or melena, foul breath, and abdominal pain. Barium enema demonstrated the fistula in 5 of 6 patients, while gastrointestinal series with barium by mouth showed it in only 3 of 7 patients.

The treatment of gastrojejunocolic fistula consists of surgical excision of the fistula, with restoration of bowel continuity. After such treatment, however, the liability of the patient to develop a recurrent peptic ulcer is high, estimated at 20 to 40 per cent. The patient should therefore be later subjected to a subtotal gastrectomy, or to vagotomy. The authors report the first case in which transthoracic vagotomy was used, successfully, in the treatment of a duodenal ulcer recurrent after excision of a gastrojejunocolic fistula.—*Henry P. Brean, M.D.*

McCREADY, F. J., BARGEN, J. A., DOCKERTY, M. B., and WAUGH, J. M. Involvement of the ileum in chronic ulcerative colitis. *New England J. Med.*, Jan. 27, 1949, *240*, 119–127.

In a series of 103 specimens of bowel from patients with chronic ulcerative colitis at the Mayo Clinic during the years 1935 through 1946, 29, or 28 per cent, showed ileal involvement. The incidence of ileal involvement was approximately the same in the 22 surgical specimens as in the 81 autopsy specimens. The process in the ileum is destructive and denuding, but is readily distinguished from regional enteritis since the bowel wall is not thickened and the lumen not stenosed.

In 22 of the 29 cases, the mucosa of the terminal ileum was diffusely denuded in what appeared to be a direct extension of the process in the neighboring colon across the ileocecal valve. The other 7 showed separate ulcers, 2 mm. to 2 cm. in diameter, with the intervening mucosa apparently normal. In all 29, the ileal process was milder than that in the corresponding colon. Five of the 29 showed perforation through the serosa.

Nineteen of the cases were examined by barium enema. In 10, ileal involvement could be recognized. The average length of the segment demonstrated to be abnormal was 30 cm. In all, the process was continuous with involvement in the diseased colon.

The authors believe that ileal involvement has important clinical significance, and that it is not simply a terminal event in intractable colitis. In 9 of the 29 cases, ileal lesions were known to be present months or years before death. Ileal involvement has occurred both in cases of fulminating disease and in long-standing chronic colitis. The authors suggest that the well known high mortality following ileostomy in ulcerative colitis may be, at least in part, the result of pre-existing ulcerative involvement of the ileostomy site.—*Henry P. Brean, M.D.*

PORTIS, SIDNEY A. Idiopathic ulcerative colitis. *J.A.M.A.*, Jan. 22, 1949, *139*, 208–214.

Because this disease begins in the rectum and sigmoid and spreads upward involving all areas of the mucous membrane so diffusely, the author became interested in studying the factors which upset the normal protective mechanisms. He describes the innervation of the bowel and outlines the nervous pathways for the transmission of emotional stimuli. In the early stages of the disease the changes are limited to that portion of the colon innervated by the sacropelvic nerves. Lysozyme (a mucolytic enzyme) is partly influenced by nervous con-

trol. It is secreted in increased amounts under stimulation of the sacropelvic nerves and the resultant loss of mucus protection allows increased activity of pancreatic enzymes in the large bowel and bacteria. With this in mind the psychiatric approach to the therapy of ulcerative colitis seems logical. The author outlines medical and psychiatric management to eradicate the mental causes and to protect the mucous membrane from enzyme activity.—*Arthur J. Present, M.D.*

Saphir, William. Abdominal apoplexy. *Am. J. Digest. Dis.*, Dec., 1948, *15*, 408.

A case of abdominal apoplexy is reported and added to the few described in the literature. The underlying pathology was mesenteric arteriosclerosis. During the observation period of twelve years the clinical symptomatology was indistinguishable from that of the "irritable colon" syndrome. Mesenteric arteriosclerosis should seriously be considered in the etiology and the differential diagnosis of the irritable colon syndrome.—*Franz J. Lust, M.D.*

Smyth, Michael J. Congenital absence of the gallbladder. *Lancet.*, Feb. 19, 1949, *1*, 301–303.

This case is thought by the author to be the first in which cholangiography has been used to confirm the operative diagnosis of congenital absence of the gallbladder.

Congenital absence of the gallbladder is not as rare as is commonly believed. At least 79 cases have been reported in the literature.

This is the case of a seventy-seven year old female who had an exploratory laparotomy October 5, 1942, at which time no gallbladder was found but a stone was removed from a greatly dilated common duct. A "T" tube was placed in the common duct and a postoperative cholangiogram clearly reproduced in the article confirmed the diagnosis of congenital absence. —*J. S. Summers, M.D.*

GYNECOLOGY AND OBSTETRICS

Montgomery, John B. Malignant tumors of the ovary. *Am. J. Obst. & Gynec.*, Feb., 1948, *55*, 201–217.

Montgomery presents a study of 107 cases of tumors of the ovary. Eighty-four of these were primary ovarian carcinomas, 7 were metastatic from uterine cancer, 2 were adenocarcinomas occurring in teratomas, 10 were granulosis cell tumors, 1 was a Krukenberg's tumor, 2 were dysgerminomas, and 1 was a Brenner tumor.

The 84 patients with primary adenocarcinoma were critically analyzed. Fifty-seven per cent of these patients were between the ages of forty and sixty years. Their symptoms often occurred late in the course of their disease. The chief symptoms were pain and enlargement of the abdomen. Less common symptoms were nausea, vomiting, loss of weight, backache, and weakness. Only 5 patients had uterine bleeding which was attributed to the ovarian tumor. Operation is recommended in every patient whose physical condition would permit such surgery even though the growth were extensive. In general, it is noted that the tumors of solid consistency are more actively malignant than those which are cystic or papillary.

The entire group of cases is analyzed from, the point of view of (1) the grade of malignancy (2) histopathological type of tumor, (3) extent or operability of the lesion, and (4) the response to radiation therapy.

Operability and the grade of malignancy are the prime factors which govern the percentage of five year survivals. The papillary cysts give the most favorable prognosis while the solid or partly solid type tumors give a very poor prognosis.

Radiation therapy is recommended as an adjunct in those instances where the growth is not completely removed. The radiologist should be acquainted with the location and the gross extent of the involvement. Since radiation therapy is largely palliative, it should not be pushed to the limit of tolerance in those cases where there is extensive disease. The best statistical results from radiation therapy are in the grade 1 tumors.—*George W. Chamberlin, M.D.*

Ashton, Dorothy L. Carcinoma of the cervix complicating procidentia uteri. *Am. J. Obst. & Gynec.*, Feb., 1948, *55*, 299–302.

This is a case report of a seventy-four year old female with a prolapsed uterus of four years' duration. Physical examination revealed a fungating growth involving the prolapsed cervix. On biopsy this showed squamous carcinoma, grade 3. The patient was given external irradiation for a total tumor dose of 4,000 r. Four weeks later the protruding mass was smaller and could be reduced and held in place by a pessary. Subsequently, however, the patient had a number of severe hemorrhages from a sloughing ulcer of the posterior lip of the cervix

and she died approximately three months from the time of her first admission to the hospital.

Another case of similar nature is reported in a forty-seven year old gravida eight with procidentia of two years' duration. This patient had a biopsy diagnosis of squamous cell carcinoma of the cervix and was given 4,800 mg-hr. of radium. Three months later a vaginal hysterectomy was done. She was then seen seven years later with a small recurrence in the vagina which was subsequently treated by radium.

The author discusses briefly the incidence of carcinoma of the uterus in procidentia and notes that it is a somewhat rare condition. She recommends that the treatment be the same for carcinoma in a prolapsed uterus as it would be for carcinoma with the uterus inside the vagina. —*George W. Chamberlin, M.D.*

Graham, Ruth M., Sturgis, Somers H., and McGraw, John. A comparison of the accuracy in diagnosis of the vaginal smear and the biopsy in carcinoma of the cervix. *Am. J. Obst. & Gynec.*, Feb., 1948, *55*, 303–307.

The avowed intention of this paper is to examine the accuracy of the cytological and histopathological methods of diagnosis in a group of proved cases of cancer of the cervix. Of 181 cases of primary epidermoid carcinoma of the cervix, both the cytological and the biopsy methods of diagnosis were correct in 81.7 per cent and incorrect in only 1.7 per cent. A false negative biopsy was found in 8.8 per cent and a false negative smear in 7.7 per cent of these patients.

The authors conclude that the vaginal smear is more accurate in their experience in the diagnosis of early carcinoma of the cervix. An extremely high percentage of cases can be diagnosed accurately if the two methods are used together.—*George W. Chamberlin, M.D.*

GENITOURINARY SYSTEM

Spatt, S. D., and Grayzel, D. M. Pheochromocytoma of the adrenal medulla. *Am. J. M. Sc.*, July, 1948, *216*, 39–50.

Five cases of adrenal pheochromocytoma are presented. These include (1) malignant type with metastases, (2) benign tumor with hypertension (one case paroxysmal and one sustained), (3) benign tumor with no signs or symptoms during life, found accidentally at autopsy.

The malignant variety is very rare, the case in this series being the ninth one on record and the only one with cerebral metastases. The earliest signs of involvement in this group are those of metastatic disease and moreover, since the histopathologic picture of the benign and malignant types is similar, metastatic disease is the only true criterion of malignancy. The absence of hypertension in those patients in the malignant group is unexplained.

The benign group with paroxysmal hypertension is fairly well recognized but, in those cases with sustained hypertension, the diagnosis must be kept in mind and careful study should be undertaken. In the latter regard, the Roth-Kvale histamine test or the benzodioxane test may be important.

The symptomless lesions do not appear to be precursors of those with symptoms (judging from the comparative ages of patients considered) and the presence of symptoms appears to be independent of the size of the lesion.—*Russell R. Jauernig, M.D.*

Gasul, Benjamin M., Glasser, Julius M., and Grossman, Aaron. Extreme hypertension in a child cured by nephrectomy. *J.A.M.A.*, Jan. 29, 1949, *139*, 305–307.

The authors present a case of extreme hypertension in a nine year old girl due to unilateral atrophic pyelonephritis. Nephrectomy was performed and the blood pressure, the urine, and chemical content of the blood returned to normal and had remained such for twenty-four months following the operation. The pronounced hemorrhagic retinitis noted before the operation was no longer present at the time of last examination. The authors then review the experimental work of Goldblatt and co-workers on this subject, and the literature. They find only 22 cases of hypertension in adults in which blood pressure has remained normal one or more years following operation. In children only 9 cases found in which the blood pressure had remained at a normal level for at least one year following the operation.

The authors point out that every case of hypertension in a child should be carefully studied and if a unilateral kidney involvement is found with the other kidney normal or showing compensatory hypertrophy, an immediate nephrectomy should be performed.—*Robert D. Moreton, M.D.*

Gibson, T. E. Lymphosarcoma of the kidney. *J. Urol.*, Dec., 1948, *60*, 838–854.

Lymphosarcoma may simulate primary renal

involvement when affecting the kidney. The author reports 3 cases of lymphosarcoma in which renal symptoms predominated.

The first case was that of a thirty-one year old white male who had had recurrent attacks of sharp pain in the left loin for six months. Retrograde pyelograms showed lateral and forward displacement of the left ureter and bulging of the upper calices of the left kidney. The pain continued and the patient lost 17 pounds. A renal sympathectomy was performed at which time a psoas muscle abscess was found. The pain and weight loss continued and a laparotomy performed one year after onset of symptoms revealed a hard mass in the left mid-abdomen which was adherent to the kidney and histopathologically proved to be lymphosarcoma.

The second case was a sixty-eight year old white male who had a six weeks' history of pains in the left flank and a 16 pound weight loss. Pyelograms showed a markedly enlarged left kidney with caliceal distortion typical of malignancy. A left nephrectomy revealed a retroperitoneal tumor which surrounded and involved the kidney and proved to be a lymphosarcoma.

The third case was a white male sixty-five years old who complained of left loin pain and burning of urination of four weeks' duration. A mass had been present in the left side of the abdomen for several months. Pyelograms were reported as showing bilateral reduplication of the pelvis of a horseshoe kidney with a large calculus. Autopsy revealed a non-fused kidney with an extensive infiltrating mass, which was lymphosarcoma histopathologically.

Clinically cases of malignant lymphomas may be divided into three groups: (1) those with symptoms of renal colic, due to constriction of the ureter by lymphomatous tissue; (2) those with kidney involvement suggesting primary renal malignancy; and (3) those in which primary retroperitoneal sarcoma is suggested with secondary renal involvement.

Fifty-one case reports of lymphoblastoma, occurring in a ten year period at the Massachusetts General Hospital, showed 64.7 per cent with urinary tract involvement. The terminology and classification of malignant lymphomas is discussed by the author.

Involvement of the kidneys by lymphoblastoma is by metastasis and by direct extension. Primary lymphosarcoma of the kidney is practically nonexistent.

The author describes 4 types of renal involvement by lymphosarcoma:

1. Single metastatic nodule.
2. Multiple metastatic nodules.
3. Capsular, in which the kidney is "engulfed as by a flow of lava."
4. Diffuse parenchymal infiltration resulting in enlargement of the kidney without gross distortion of the architecture.

The author includes illustrative drawings to illustrate the above types.

Symptoms include pruritus, intermittent fever of the Pel-Ebstein type, vague abdominal symptoms, back pain, anemia, weight loss, purpuric manifestations, superficial lymphadenopathy, etc.; the blood picture may or may not be leukemic. The onset is insidious. Acute inflammatory lymphadenopathy must be excluded. Most cases must depend on biopsy for diagnosis.

Nephrectomy has been performed in a few cases of malignant lymphoma with renal involvement without benefit or cure. The latter could not be expected in view of the disseminated nature of the disease. A significant number of cases have been reported in the literature where localized lymphosarcoma, confined to a single kidney, has been subjected to surgical removal and irradiation. Hilling, in a series of 130 patients, found 24.5 per cent five year survival, and 10 per cent eight year survival; 17.2 per cent of the series were apparently cured. Malignant lymphoma is very radiosensitive. Irradiation has a valuable palliative effect. More recently nitrogen mustard therapy has been tried with results similar to those of irradiation.—*Rolfe M. Harvey, M.D.*

Hackworth, L. E. Urethrography in infants and children. *J. Urol.*, Dec., 1948, *60*, 947–951.

The author employs the contrast medium visco-rayopake as a medium of choice in urethrography of infants and children. A modification of the medium, called rayopake, is now available and is less dense, making it possible to visualize small polyps.

Three to 10 cc. of the medium is injected and a film exposed during the injection, which should be slow and steady. The exposure should be for 1/60 sec. at 40 kv. and 400 ma. using a Lysholm grid. Films are exposed in the anteroposterior and right oblique projections.

The author illustrates his article with normal urethrograms of patients from birth to the age

of twelve. A few examples of pathological conditions are also illustrated.—*Rolfe M. Harvey, M.D.*

Diehl, William K., and Hundley, J. Mason. Urinary tract changes in cervical carcinoma. *Surg., Gynec. & Obst.*, Dec., 1948, *87*, 705–715.

Thirty-seven patients comprise the series studied. The status of the urinary tract was determined before and after radiation therapy.

Conclusions

1. The more advanced the original cervical carcinoma, the more frequent is the association of urinary tract dilatation before the initiation of radiation therapy.

2. The development of a hydronephrosis and hydroureter during or after treatment has in every case been associated with advancing malignancy and has invariably proved to be a grave prognostic sign.

3. The majority of patients with carcinoma grades 1 and 2 did not show changes in the urinary tract before therapy and none of the patients in these groups developed a dilatation during the period of time between examinations.

4. Urological studies of all patients who have cervical carcinoma are indispensable for purposes of intelligent evaluation and management.

5. Radiation therapy will not cause stricture of the ureter if the dosage is not excessive and provided the source of the ray is displaced from the ureter by accurate maximal vaginal packing.

6. Each patient in this series received a total of 6,000 milligram-hours of radiation (in two doses of 3,000 milligram-hours each with a three week interval). Three weeks later a course of deep roentgen therapy totaling 6,000 to 8,000 roentgens was administered. Twelve weeks later a second cycle of deep roentgen therapy was given.—*Mary Frances Vastine, M.D.*

Albers, Donald D., McDonald, John R., and Thompson, Gershom J. Carcinoma cells in prostatic secretions. *J.A.M.A.*, Jan. 29, 1949, *139*, 299–303.

This study was undertaken to prove that carcinoma of the prostate can be diagnosed from a microscopic examination of prostatic secretion.

The cases studied were considered in two groups. The first group consisted of 100 cases in which a preoperative diagnosis of non-malignant hypertrophy of the prostate had been made.

In the prostatic secretions of each of 3 of these cases, cells and clumps of cells were found which showed characteristics of malignant neoplasm. The diagnosis of malignant neoplasm in these 3 cases was confirmed by histopathological examination of tissues removed by transurethral resection.

In a fourth case malignant disease was found in the resected prostate, although no characteristic cells had been discovered in the secretions preoperatively.

The second group consisted of 41 cases in which a preoperative diagnosis of malignant disease of the prostate had been made. In 17 of these, secretions could not be obtained. Of the remainder there were 13 positive and 3 negative cases in which the preoperative examination of the secretion agreed with the postoperative examination of tissues. Six of the cases in this series were not operated upon. In the remaining 2 cases there was disagreement between the results of examination of the secretion and the histopathological tissue studies.—*I. Schwartz, M.D.*

SKELETAL SYSTEM

Lavalle, L. I., and Hamm, F. C. Osteitis pubis; its diagnosis and treatment. *J. Urol.*, Jan., 1949, *61*, 83–95.

Osteitis pubis is an inflammatory disease of the pubic bone following suprapubic incisions for prostatectomy or urinary bladder conditions. Under expectant treatment the disease is self-limited although usually three to twelve months of prolonged pain and disability occurs. The literature is reviewed by the authors who report 7 additional cases and present a new method of therapy which results in rapid amelioration of symptoms.

The onset of the disease follows operation by three to seven weeks. The onset is sudden with excruciating pain over the symphysis extending to involve the rami of the pubic bones. Locomotion is difficult because of pain. Pain is induced by walking, sitting up, passive spreading of the thighs, and stretching of the adductor muscles. A low-grade fever is present without localized pubic swelling or redness. The etiology is obscure. The first pathological change is a periostitis of the pubic bone which is followed by cartilage destruction of the symphysis and resultant separation of the pubic bones.

Roentgenographic corroboration of the osteitis was not obtainable until one to nine weeks following onset of symptoms. A fuzziness of the periosteum was first noticed. Within two weeks the pubic rami became moth-eaten, simulating metastatic malignancy.

The authors found treatment with intramuscular vitamin B complex and diathermy of great value causing rapid regression of symptoms. In one case in which the above therapy was ineffective, roentgen therapy proved effective. The authors suggest trying B complex first and reserving roentgen therapy for refractory cases. They report their 7 cases in detail and include illustrative roentgenograms. Roentgenograms should be made at weekly intervals.—*Rolfe M. Harvey, M.D.*

SCHACKELFORD, RICHARD T., and BROWN, WEBSTER H. Restricted jaw motion due to osteochondroma of the coronoid process. *J. Bone & Joint Surg.*, Jan., 1949, *31-A*, 107–114.

In 1943, the authors reported 2 cases of osteochondroma of the coronoid process of the mandible. They pointed out that the tumor had been an unrecognized cause of progressive limitation of motion of the jaw, and that its recognition by roentgenograms taken in the usual position employed for studies of the mandible was unlikely. In this paper they report a follow-up of the original 2 cases, and record 2 additional cases and again call attention to the condition and the means of its recognition and correction.

Important points to emphasize in the diagnosis of the condition are a slowly progressive, painless limitation of movement of the jaw; unilateral occlusion of the teeth; a bony-hard swelling in the region of the zygoma on the affected side; and a bony projection visualized by the roentgenograms in a special position. The important point in the roentgenographic technique is to have the central ray directed tangentially to the cheek of the affected side, instead of being centered on the midline symphysis of the chin. The anteroposterior position usually employed for demonstrating the nasal sinuses may also afford a good view of the lesion.

The fourth case reported differed from the others in that the tumor was osseous rather than an osteochondroma. It arose from the zygoma instead of the coronoid and followed a definite history of trauma with roentgenographic evidence of previous fracture of the zygoma. The authors included it in the report, however, because it caused, due to its impingement on the coronoid process, the same symptoms of progressive limitation of the jaw.

Treatment consisted of surgical excision. The operative technique is described in detail and it has proved to be satisfactory.—*R. S. Bromer, M.D.*

THOMPSON, J. E. M., and TANNER, FRANK H. Tumoral calcinosis. *J. Bone & Joint Surg.*, Jan., 1949, *31-A*, 132–140.

Three cases of tumoral calcinosis are reported in this paper. The authors describe a third type of calcinosis, the tumoral, which they add to the previously described types, calcinosis circumscripta and calcinosis universalis. Tumoral calcinosis occurs in young individuals of both sexes. There is probably a familial tendency. The tumors are large, rapidly growing to a definite, self-limited size, they usually occur in or near large joints, bursal sites and in connective tissue, immediately overlying bone and muscle attachments. The tumors are hard and lobulated, have fairly well-defined limitations in contour, are attached firmly to the underlying deep structures, and may show some infiltration into the muscles. There is usually no pain, tenderness, or limitation of joint motion. There is a definite multilocular, cystic arrangement of the tumor, which has thick rubbery walls, showing inflammatory foreign body, giant cell reaction. Deep cystic areas are filled with calcareous material and some milky fluid. Surgical removal relieves the condition. Drainage and secondary infection, which occurred in one of their cases, apparently aggravate metastatic calcification and generalized amyloidosis may develop.

The 3 patients who are reported in this paper were brothers. The sites of the tumors, as well as the microscopic appearance and laboratory data in the 3 cases, were similar. The tumors appeared during adolescence and were self-limited in size, after growing for about one year. Most of the tumors arose near joints in the region of gliding surfaces or bursae, but extended into muscles. The tumors were fairly well defined, lobulated, and fluctuating. They were not accompanied by pain, tenderness, or limitation of motion. Complete removal seems to cure the condition locally, but similar tumors may appear elsewhere. The fatal ending in one case was the result of long-standing infection, due to early secondary infection following

drainage, amyloid changes in the viscera, and unfortunate circumstances accompanying his critical condition when final drainage was attempted. Roentgenograms of 2 of the cases are reproduced.—*R. S. Bromer, M.D.*

Wilson, John C., and McKeever, Francis M. Traumatic posterior (retroglenoid) dislocation of the humerus. *J. Bone & Joint Surg.*, Jan., 1949, *31-A*, 160–172.

Eleven patients with posterior dislocation of the shoulder were studied by the authors. One of these suffered a bilateral posterior dislocation, making a total of 12 shoulders in which the head of the humerus was displaced to the retroglenoid position. The cause of the dislocation in 7 of the patients was external trauma. In 3 of them the cause was an epileptic convulsion, one having a bilateral dislocation. One patient suffered a luxation as a result of violent muscle contraction initiated by contact with a live electric wire which suggests that this type of dislocation might be an occasional complication of electric-shock therapy. In 8 of the dislocated shoulders there was concomitant fracture.

In roentgenograms of the posterior dislocations of the shoulder, the humerus is in extreme internal rotation. Loss of the sharp outline of the greater tuberosity and the disappearance of the half-moon shadow of the superimposed glenoid and humerus have been described as useful signs. Attention has been called to the fact that, in a posterior dislocation, the lesser tuberosity of the humerus forms the medial border of the roentgenographic shadow of the humeral head, and also that the lower third of the glenoid may be exposed in the anteroposterior roentgenogram of a patient with posterior dislocation. One author has stated that in an anteroposterior roentgenogram of a posterior dislocation, the greater tuberosity lay directly under and behind the coracoid process. These, the authors consider, are useful signs but they may be simulated if an anteroposterior roentgenogram is taken with the humerus in extreme internal rotation, or with the elbow supported in a Velpeau bandage.

The definite and unmistakeable diagnosis of posterior dislocation of the shoulder is made by obtaining both anteroposterior and lateral roentgenograms. In the lateral (axillary) roentgenogram of a posterior dislocation, the humeral head is seen to lie posterior to and outside of the glenoid cavity and to be widely separated from the coracoid process.

Prompt recognition and treatment of posterior dislocation of the shoulder by closed manipulation yield excellent results. Even with early prompt reduction, however, the replacement is unstable and there is danger of spontaneous redislocation. This danger, the authors believe, can be avoided by the simple expedient of tranfixing the reduced head of the humerus to the acromion process with cruciate wires. This does not require an open operation. Failure of early recognition necessitates open operative intervention. The results of late treatment are poor.—*R. S. Bromer, M.D.*

Klein, Armin, Joplin, Robert J., Reidy, John A., and Hanelin, Joseph. Roentgenographic changes in nailed slipped capital femoral epiphysis. *J. Bone & Joint Surg.*, Jan., 1949, *31-A*, 1–22.

In this paper, the authors present roentgenograms illustrating, (1) the results of the treatment of slipped capital femoral epiphysis by nailing in situ and by nailing after osteotomy and replacement; (2) the prevention of further slipping, acceleration of fusion of the epiphysis, and preservation of a relatively normal anatomy of the hip, accomplished by this method; (3) the absence of the accelerated fusion in 4 cases, in which growth persisted until the epiphyseal plate had advanced beyond the end of the nail. In the authors' series there were 31 patients, 4 with bilateral involvement, representing a total of 35 hips, nailed in situ because the amount of slipping was less than 1 centimeter. After an average follow-up period of thirty-two months, the average index of motion was 90 per cent of normal, and the average of percentage of normal hip function was 96. Roentgenograms taken at the end of a seven year follow-up period show the results which can be expected from nailing in situ when the minimal amount of slipping is detected early. Sixteen patients were reported with marked slipping, more than 1 centimeter. They were treated with arthrotomy, osteotomy through the epiphyseal plate, replacement of the head to its anatomical position in relation to the neck, and lateral nailing for fixation. Roentgenograms are shown illustrating the results after an average follow-up period of thirty-three and one-quarter months.

The results of their method of treatment suggest that adequate replacement with a minimal amount of circulatory embarrassment to the femoral head and neck, followed by early

immobilization and weight-bearing with the aid of crutches, is of paramount importance. Treatment of the slipping while it is minimal permits a simpler operative procedure with an excellent prognosis, and eliminates the hazards and the inferior results stemming from the difficult procedure which is indicated after the slipping has become marked.—*R. S. Bromer, M.D.*

KEY, J. ALBERT. Survival and growth of an epiphysis after removal and replacement. *J. Bone & Joint Surg.*, Jan., 1949, *31-A*, 150–152.

In 1946 the author reported the case of a boy eleven years of age, who had suffered a dislocation of the elbow with a fracture through the neck of the radius and displacement of the head of the radius backward into the elbow joint. He was operated upon about forty-eight hours after the injury and the head of the radius, which included the epiphyseal line and a bit of the neck, was found completely free in the posterior portion of the elbow joint. It was lifted out with forceps, placed in a folded towel, and laid upon the instrument table while a bed was prepared for it. It was then replaced on the neck and the elbow was flexed to 90 degrees. The capsule was sutured around the head, the wound was closed, and the extremity was immobilized in a plaster cast. Thirteen months after the operation it was found that the movements of the elbow were almost normal, the roentgenograms showed the head of the radius united and in satisfactory position. It was Key's opinion that the head of the radius and the epiphyseal line were living.

The patient is now fifteen years old and a little over four years has elapsed since the operation. He plays baseball and states that he is able to throw a baseball as well as other boys on his team. Flexion and extension of the elbow and rotation of the forearm are normal. There is no evidence of shortening of the radius or of deformity of the elbow or wrist. The only abnormality noted is slight soft crepitus on palpation over the head of the radius while the arm was being rotated. Roentgenograms show the head of the radius living and united with the neck in slight angulation. The epiphyseal line appears closed. The boy has an approximately normal right upper extremity.

Key believes that this report affords sufficient evidence to warrant reiteration of the statement that, in children, epiphyses should be replaced if possible, because they may survive and the bone may grow normally, even though the epiphysis has been completely separated and is without blood supply. With bone banks now existing, this case is of interest, Key states, because it brings up the perennial question of how much of a bone graft lives. It may constitute some evidence on behalf of the autogenous graft.—*R. S. Bromer, M.D.*

COMPERE, EDWARD L. Avascular necrosis of large segmental fracture fragments of the long bones. *J. Bone & Joint Surg.*, Jan., 1949, *31-A*, 47–54.

The clinical significance of traumatic devitalization of portions of the shafts of long bones has not been recognized by many fracture and orthopedic surgeons. A large fragment in a comminuted fracture of the femur, tibia, humerus, or one of the bones of the forearm, may be so detached from surrounding soft tissue that nutrient, metaphyseal, and periosteal blood vessels supplying the fragments are separated from it. In such cases, the segmental fragment and sometimes the ends of the two major fragments, are so devitalized that they are in the same situation as a full-thickness autogenous bone graft.

Five cases are reported in detail which demonstrate the typical course of a segmental fracture of a major long bone. The bone which is necrotic retains a density greater than that of the living adjacent bone. This is illustrated in the roentgenograms of the cases reported. This relative difference in density is produced by the fact that the living bone atrophies following the fracture, while the bone which is deprived of its vascularity is unable to atrophy, having insufficient blood supply to take away the opaque mineral salts

Compere believes that with reasonable certainty a marked delay in union of large segmental fractures of the mid-shaft or ends of a major long bone may be predicted. The larger the separate fragment in a segmental fracture of a long bone, the poorer the prognosis from the standpoint of viability of that fragment. In the event that a fragment is partially devitalized and does not retain enough intact blood vessels to maintain a reasonable exchange of arterial and venous blood, some of the bone cells will die. If most of these cells die, the fragment will become necrotic, although massive sequestration does not occur. Union may then be delayed for many months. With good

fixation, however, healing of the fracture will take place before the large fragment has undergone complete replacement by creeping substitution. If a principal nutrient artery remains uninjured and supplies the large segmental fragment, aseptic necrosis may not occur, and the prognosis for undelayed healing of the fracture is good.—*R. S. Bromer, M.D.*

Moore, John Royal. Delayed autogenous bone graft in the treatment of congenital pseudoarthrosis. *J. Bone & Joint Surg.*, Jan., 1949, *31-A*, 23–29.

The various theories concerning the etiology of congenital pseudoarthrosis are enumerated by Moore. The etiology still remains obscure. He cites previous case reports in the literature and quotes historical data from the study of Camurati who collected 118 cases from the literature. The histopathological characteristics of the lesion have been generally accepted as conforming to those of acquired pseudoarthrosis. Inert, sclerosed, pointed atrophic bone ends (having the appearance of sucked candy), surrounded by a sleeve of dense connective tissue and developing ultimately the characteristics of a nearthrosis including the cartilage and the joint cavity, are the principal findings in the mobile type. Moore classifies congenital pseudoarthrosis into two types:

1. Pre-pseudoarthrosis. In this stage the long bone is bowed or bowing, the apex of the bow is narrow, the medullary canal becomes obliterated, and spontaneous or induced fracture (often surgical) occurs, followed by pseudoarthrosis.

2. Pseudoarthrosis. Various degrees may occur, from simple fibrous to frank nearthrosis, including sclerosis of the bone ends, cartilage, and joint cavity.

He describes the preparation of the delayed autogenous bone graft, the preparation of the pseudoarthrosis site, the implantation of the grafts, and the after-treatment.

Eight cases are reported with clinical and laboratory data in all of which he accomplished successful osteosynthesis. In 6 of them the deformity was in the leg; in 1 in the ulna and 1 in the femur. The delayed autogenous graft is capable of stimulating osteogenesis. Bony union occurred in each instance. He emphasizes the importance of the continuation or primary immobilization until the medullary cavity has been formed. A bimonthly roentgenographic check-up is essential during the first two years after operation. Moore feels the follow-up period in all, of his cases is probably too short to justify final conclusions. The fact that union occurred in every case in which the delayed graft was used, including those cases in which re-operation was done, is regarded as distinctly encouraging and points to the effectiveness of this type of graft. The treatment of congenital pseudoarthrosis will probably never be entirely effective until the etiology has been determined.—*R. S. Bromer, M.D.*

Patrick, James. Intracapsular fractures of the femur treated with a combined Smith-Petersen nail and fibular graft. *J. Bone & Joint Surg.*, Jan., 1949, *31-A*, 67–80.

Patrick believes that in the treatment of intracapsular fractures, the use of a fibular graft with a trifin nail does not increase the mortality, and the addition of the graft lengthens the operation time by only a few minutes. In his small series of cases a much higher proportion of bony union occurred and fewer cases of aseptic necrosis appeared as compared with the published results of cases treated with a nail alone.

In one patient, aseptic necrosis developed in spite of non-weight-bearing for a period of seventeen months. He believes it is evident that lack of weight-bearing does not prevent necrosis, although it may minimize collapse of the head and it may be of some value in delaying the onset of osteoarthritis. Early weight-bearing may result in further impaction and shortening of the neck and this may cause the graft to penetrate the joint. For this reason weight-bearing should be avoided for the first three months, and the fibular graft should be inserted a little less than the depth of the trifin nail.—*R. S. Bromer, M.D.*

Phemister, Dallas B. Treatment of the necrotic head of the femur in adults. *J. Bone & Joint Surg.*, Jan., 1949, *31-A*, 55–66.

In this paper 2 cases of necrosis of the head of the femur, one associated with an ununited fracture of the neck and the other with an ununited fracture at the junction of the head and neck, are reported. In the second case, the fracture developed after healing of a primary fracture located distally in the neck. They were treated by drilling two holes across the neck and upper portion of the head and by insertion of a rectangular bone graft into each hole. In a third case, also reported, of ununited fracture

with death of the head of two years' duration, the lower bone graft was inserted across the fracture line deep into the head, while the upper graft was inserted only to the margin of the upper portion of the head.

Phemister states the cases present evidence that drilling and bone-pegging of the upper portion of the necrotic head of the femur which has undergone little or no collapse may increase the rate of transformation of the structure into living bone, shorten the non-weight-bearing period, and decrease the incidence of collapse, deformity, degenerative arthritis and poor functional results.—*R. S. Bromer, M.D.*

Babb, Frank Shaleen, Ghormley, Ralph K., and Chatterton, Carl C. Congenital coxa vara. *J. Bone & Joint Surg.*, Jan., 1949, *31-A*, 115–131.

Congenital coxa vara is an infrequent deformity of the femoral neck in children, recognized since 1896 and characterized by a defect which pathologically and roentgenologically is not unlike that seen in aseptic necrosis. Except for its frequent association with a short femur, congenital coxa vara seems to occur entirely apart from other congenital deformities and to possess sufficiently distinguishing characteristics to warrant recognition of it as a separate entity.

In this paper, the authors summarize the current concepts of the deformity and differentiate it from multiple congenital deformities and they, in addition, report 15 cases. They state that unless one is familiar with the condition, the correct roentgen diagnosis is usually not made. The femoral neck is observed at once in the roentgenogram to be bent, so that the head is depressed and the distal part of the limb is thereby adducted. The epiphyseal line for the head is more vertical than it is normally, and it appears to be branched like an inverted Y. It is not, however, a branching of the epiphyseal line, but a "disorganized segment," sometimes referred to as the vertical fissure, in which an abnormal ossification process has resulted in a defect not unlike that seen in aseptic necrosis or localized osteochondritis. The greater trochanter is elevated and may have a peculiar beaked appearance. The femoral head is comparatively large, somewhat translucent and lies in the bottom of the acetabulum. The acetabulum may be deformed in outline, shallow and defective inferiorly.

There is still no universal agreement as to the cause of congenital coxa vara. The authors state that the modern tendency to explain it as an aseptic necrosis makes it all the more essential that the condition be separated from other obvious congenital deformities. They attempt to demonstrate pathologically that the coxa vara occurring as one of multiple congenital deformities is not the same as this infantile lesion, the cause of which may be even postnatal.

The lesion is to be suspected in any child who is born with an obviously short leg or who limps when walking is first attempted. The findings on physical examination suggest a congenital dislocation of the hip, with shortening, high position of the trochanter, and a positive Trendelenburg test. However, there is no telescoping and the femoral head is still palpable beneath the femoral vessels at the groin. The roentgenogram will confirm the diagnosis if the lesion is not mistaken for an ununited fracture of the femoral head, a rare condition in children.

The authors regard congenital coxa vara, as they define it, as a not proved congenital deformity. The frequent association of the lesion with a short femur is probably due to a common etiological factor, possibly avascular necrosis. Coxa vara associated with multiple congenital deformities is not the same lesion as the one with which they are concerned in this paper. Subtrochanteric osteotomy at about six to eight years of age, with wide abduction of the distal part of the limb, is the treatment of choice.—*R. S. Bromer, M.D.*

Johnson, E. W., and Janes, Joseph M. Metastatic adenocarcinoma of the talus from the rectosigmoid region. *J. Bone & Joint Surg.*, Jan., 1949, *31-A*, 181–184.

Reports of metastases to an extremity in cases of carcinoma of the rectum are exceedingly rare. A case is reported in this paper, a male, aged thirty-seven, who was operated upon for adenocarcinoma of the rectum. Approximately five years later, he developed intermittent pain and swelling of the left leg and ankle. Roentgenograms of the left ankle showed a destructive process of the posterior superior aspect of the talus. A specimen was removed, the histopathological sections showing adenocarcinoma. There was also evidence of metastatic involvement of the lungs in the roentgenograms of the chest.

The anatomical pathways involved in the

spread of rectal carcinoma are the classical ones, namely (1) direct extension, (2) the lymph vessels, (3) the blood stream, (4) transplantation through the peritoneal cavity. In spite of all that has been written on metastatic growth of tumors, not one of the theories explains the scarcity of metastatic involvement below the knees and elbows. It might be conjectured that the more proximal metastatic lesions cause death before evidence of metastasis to an extremity is evident. It has also been postulated that secondary tumor growths below the knees and elbows are rare because of the decreased temperature of the extremities. Whatever the cause, reports of metastases to the extremities from carcinoma of the rectum are very rare. Weston reviewed the literature and found only 6 cases reported.—*R. S. Bromer, M.D.*

Schlesinger, Philip T., Keats, Sidney, and Ruoff, Andrew C. Fibrous dysplasia. *J. Bone & Joint Surg.*, Jan., 1949, *31-A*, 187–191.

A case of fibrous dysplasia of the tibia is reported which presented the unusual complication of massive hemorrhage with absorption of a large section of the tibia. The patient showed none of the extraskeletal manifestations of Albright's disease. Because of the long duration of the case and the fear that sarcomatous degeneration had occurred, an amputation of the leg, supracondylar of the right thigh, was performed. The microscopic picture revealed not only the characteristic findings of fibrous dysplasia but also evidence of fairly recent massive hemorrhage and degeneration of tissue.

The authors believe on the basis of their case and also on a similar one reported by Jaffe, that the clinical picture of fibrous dysplasia should be broadened to include the complication of massive hemorrhage. This complication may be suspected in a patient with fibrous dysplasia, who presents a rapidly expanding bone tumor.—*R. S. Bromer, M.D.*

Falconer, Ernest H., and Leonard, Maurice E. Skeletal lesions in Hodgkin's disease. *Ann. Int. Med.*, Dec., 1948, *29*, 1115–1131.

It was formerly believed that Hodgkin's lymphogranuloma spread only through lymphatic tissue. At present the conception that the lesions of Hodgkin's disease may also spread through the reticuloendothelial system appears to be quite well established. Some investigators believe this disease should be considered as a disease of the reticuloendothelial system.

There appears to be quite general agreement among investigators of Hodgkin's disease that skeletal involvement means marrow involvement. The actual mechanism of marrow involvement has not been adequately demonstrated. Three methods are commonly mentioned: direct invasion from adjoining granulomatous lesions, usually lymph nodes, hematogenous invasion through small lymphogranulomatous emboli, or transference of an agent capable of initiating a focus in situ. This latter method could also initiate a lesion primary in the bone marrow, bearing in mind, of course, that the etiology of Hodgkin's disease is at present unknown. Several authors have reported cases where the first clinical evidence of the disease was demonstrated in the skeleton.

Twenty cases of Hodgkin's disease proved by autopsy were investigated for evidence of marrow involvement. The specimens of marrow studied were the usual routine specimens taken at autopsy either from the sternum or from a rib.

Two types of investigation are recorded in the statistics of this table: namely roentgenologic and postmortem. A comparison of these percentages derived from the two types of study indicate that roentgenologic statistics may be misleading unless the limitations of this method of study are kept in mind. Roentgenologic bone surveys must be carried out on all patients studied and roentgenograms carefully searched for skeletal lesions in order to obtain the true statistical incidence. Critical study of postmortem marrow investigations reveals a similar type of statistical error.

Skeletal lesions are practically synonymous with marrow lesions, the focus originating in the bone marrow and enlarging until it erodes the contiguous cortex of the bone. Occasionally instances are encountered where the granuloma appears to invade the bone from without, entering through the periosteum from contiguous lymphogranulomatous tissue. It is the authors' impression that when this occurs the disease is widespread and fairly well advanced.

It seems reasonable to assume that the marrow and the spleen because of their histological structure may be quite as favorable sites for early proliferation of Hodgkin's lymphogranuloma as lymph nodes. Both marrow and splenic tissue have labile growth potentialities in their cellular components, and their filter propensities with stasis of blood in the sinusoids, might

readily harbor the agent responsible for proliferation of lymphogranulomatous lesions.

The authors believe the incidence of skeletal lesions shown by roentgenologic examination is necessarily incomplete and too low, as marrows with Hodgkin's lymphogranuloma may contain lesions too small to involve the adjacent cortical portion of the bone.—*Eugene J. McDonald, M.D.*

Piedmonte, M., and Sirtori, C. Osservagioni sul confronto fra il quadro anatomico e il quadro radiologico dei tumori giganto-cellulari delle ossa. (Observation on the comparison between the anatomical and roentgenological appearance of giant cell tumors of bone.) *Radiol. med.*, Dec., 1948, *34*, 809–822.

The authors are of the opinion that benign giant cell tumors never become malignant nor give rise to distant metastases. In support of this view they state that in those cases reported of malignant giant cell tumors with pulmonary metastases, the histopathological structure has always been that of a sarcoma rather than of giant cell tumor.

They review some of their cases, of their total collection of 18, to demonstrate that benign giant cell tumors may invade locally surrounding tissues and break through the bone cortex. The roentgen findings in these cases may be interpreted as those of a malignant bone tumor. The cellular structure, however, retains all the characteristics of a benign giant cell tumor. These tumors, they believe, remain and behave as benign growths.

In another case they report in detail, they attempt to demonstrate that an osteogenic sarcoma may be mistaken for a benign giant cell tumor histopathologically because the sarcomatous element may be so scanty in certain parts of the tumor that it may be overlooked. The roentgen findings in their case suggested a primary malignant bone tumor. From the tissue examined after extensive curettement of the tumor located in the proximal end of the tibia, a microscopic diagnosis of benign giant cell tumor was made. The area was treated by irradiation, giving a tumor dose of 3,600 r, delivered through four portals. Two months later, amputation of the extremity became necessary and re-examination of tissue removed from the same lesion again resembled a giant cell tumor, except in scattered areas where sarcomatous element could be detected. Within a year the patient developed distant metastases and died.

Thus the authors show that there may be discrepancy between the roentgenological appearance and the histopathological structure of benign giant cell tumors and that in each case all factors should be evaluated after microscopic examination of the tumor tissue.—*Peter E. Russo, M.D.*

BLOOD AND LYMPH SYSTEM

Freeman, Norman E., and Miller, Earl R. Retrograde arteriography in the diagnosis of cardiovascular lesions. I. Visualization of aneurysms and peripheral arteries. *Ann. Int. Med.*, Feb., 1949, *30*, 330–342.

Recent advances in vascular surgery have created a demand for more accurate methods of diagnosis. Just as developments in surgery of the abdomen, urinary tract, head, and thorax have been aided by improvements in roentgenographic technique, so advances in vascular surgery may be expected to develop with visualization of the heart and blood vessels after the injection of contrast media.

Visualization of aneurysms of the thoracic aorta and innominate artery was attempted on three occasions but the results were not entirely satisfactory, presumably because of the difficulty of introducing a sufficient quantity of radiopaque material rapidly.

The authors have not been successful in attempts at visualization of the abdominal aorta except in the patient in whom the catheter was introduced through a cannula inserted into the femoral artery. In animal experiments Fariñas showed fluoroscopically that to be successful the injection must first overcome the inertia of the blood flow and, second, must overcome the blood pressure. It may be that injection of a larger quantity of solution under higher pressure would have yielded more satisfactory results. The danger of thrombosis at the site of injection, especially where a large needle is inserted into a small artery, is very real. With the use of 70 per cent diodrast, the possibility of serious systemic reaction should be considered, especially when there is any opportunity for the contrast medium to enter the cerebral circulation. Attention is called to the complications which may result from the use of this method.—*Eugene J. McDonald, M.D.*

Meyer, Andrew H., and Overmiller, W. C. The use of a nitrogen mustard in Hodgkin's disease and lymphosarcoma. *Ann. Int. Med.*, Feb., 1949, *30*, 381–386.

Investigation into the mode of action of mustard gas revealed a type of action of sulfur and nitrogen mustards which is unlike that of any other chemical agent and resembles in many ways that of roentgen rays. The effects of the mustards on lymphoid tissue, coupled with the finding that actively proliferating cells are selectively vulnerable to the cytotoxic action of the mustards, led to its use in the treatment of neoplasms of lymphoid tissue. The amount recommended as a single course is 0.1 mg. per kilogram intravenously on four successive days (a total of 0.4 mg. per kilogram). Subsequent therapy varies with each patient. Nitrogen mustard must be administered only by the intravenous route, taking great care to prevent extravasation of the solution.

Two cases of Hodgkin's disease, treated with nitrogen mustard, methylbis (b-chloroethyl) amine hydrochloride, responded favorably at a period when further roentgen therapy was not considered advisable. Of the 3 cases of lymphosarcoma, treated after further roentgen therapy was not deemed advisable, 1 is leading a useful home life eight months after initial therapy; 1 received marked subjective and objective relief but died in her sleep one mouth after the last course of therapy, and the third experienced considerable relief from pain, disappearance of peripheral lymphadenopathy, but no relief from dysphagia or vomiting. Autopsy revealed metastatic tumor nodules in the liver and kidneys.—*Eugene J. McDonald, M.D.*

GENERAL

Bass, H. E., Schomer, A., and Berke, R. Coccidioidomycosis. *Arch. Int. Med.*, Dec., 1948, *82*, 519–528.

It is known that about 80 per cent of the long time residents of the endemic area in the southwestern desert area of this country are infected and that about 25 per cent of the military personnel with several months' residence in the endemic area became infected. From 20 to 25 per cent of infections are clinically recognizable.

The primary purpose is to present data on a series of patients residing in New York City with protracted residual pulmonary lesions of coccidioidomycosis and to discuss the pitfalls in diagnosis.

The residual lesions were of all types, i.e., nodular densities, cavities, mottled infiltrations, fibrosis, pleural effusion and calcification. These residual pulmonary lesions characteristically showed little or no change over a period of observation of from two to five years. All patients gave a history of residence in the endemic area, varying from three months to two years. Skin sensitivity to coccidioidin was observed to diminish with the lapse of time. However, Smith and associates asserted that sensitivity to coccidioidin is durable and that, although it may wane, its loss is usually slow. Thus, while a dilution of 1:1,000 commonly yields strong reactions in early cases, it was found preferable to employ 1:100 and 1:10 dilutions in residual cases. All the patients in this series reacted to coccidioidin in 1:100 dilution. The reactions were usually of a mild type (1 or 2 plus).

A negative reaction to the coccidioidin skin test was misleading in the presence of a disseminating lesion. In such an instance the failure to react to coccidioidin was presumed to be related to energy. However, serologic examination is usually diagnostic and can often be confirmed by recovery of the spherules in a tissue biopsy, from sputum or from draining sinuses. In cases of cavity formation, no evidence of bronchogenic spread or of seeding to other parts of the lung fields, such as occurs commonly in tuberculosis, was observed.

The resemblance of the residual pulmonary lesions to those in tuberculosis was striking. In several of the cases, a diagnosis of tuberculosis had been made after the patient's return to civilian life, usually after a routine chest survey or pre-employment roentgenoscopic examination. In a number of cases, a positive reaction to both the tuberculin and the coccidioidin skin test made diagnosis difficult. In such cases a presumptive diagnosis of coccidioidomycosis was made on the basis of the following evidence: (*a*) history of exposure in an endemic area; (*b*) roentgenographic evidence of a pulmonary lesion, usually a solitary cavity or nodular density, which showed no change after months of observation; (*c*) indication of an identical lesion in the roentgenogram of the chest taken on separation from military service; (*d*) a positive reaction to the coccidioidin skin test, and (*e*) absence of tubercle bacilli. Almost

all the patients observed were asymptomatic.—*Eugene J. McDonald, M. D.*

McANALLY, ALBERT K., and DOCKERTY, MALCOLM B. Carcinoma developing in chronic draining cutaneous sinuses and fistulas. *Surg., Gynec. & Obst.*, Jan., 1949, *88*, 87–96.

The authors wish to emphasize the occurrence of carcinoma in chronic inflammatory sinuses for the following reasons: (1) The development of malignancy on this basis is not particularly rare. (2) The diagnosis can be made clinically at a relatively early stage. (3) The neoplasms are, in general, slow-growing and can be treated successfully if diagnosed during the long latent period which usually precedes the stage of metastasis.

Conclusions

1. Malignant changes in a chronic osteomyelitic sinus should be suspected when a growth appears in the sinus, the discharge becomes more foul-smelling or hemorrhagic, a painful swelling appears and when the roentgenogram reveals a pathologic fracture or areas of rarefaction surrounding the osteomyelitic cavity. There were 9 such cases in the author's series.

2. In this series there were 2 cases in which metastasis occurred from a malignant lesion in a chronic osteomyelitic sinus; these added to the 9 cases reported in the literature make 11 cases in which distant spread occurred.

3. It is proposed that the regional lymph nodes be treated with roentgen rays and that even a prophylactic dissection of these nodes be considered.

4. The transitional epithelium from the erratic glands of the anal crypts probably invades the fistula in ano tracts and this may account for the fact that acini are located in some of the fistulous tracts which have shown malignant change. Three such cases are presented in this series.

5. Malignant disease occurring in a chronic empyemic sinus is a surgical rarity. There was one in this series

6. When the biopsy reveals epithelial hyperplasia, the sinus or fistula should be excised and blocks studied for evidence of malignant changes. If neoplastic tissue is found, amputation should be done in cases of osteomyelitis and rather radical excision should be done for fistula in ano and empyemic sinuses.—*Mary Frances Vastine, M.D.*

RADIOISOTOPES

CRISTOL, D. S., BOTHE, A. E., and GROTZINGER, P. W. Radioactivity and urinary tract calculi. *New England J. Med.*, Sept. 16, 1948, *239*, 427–429.

A patient with a vesical calculus, prostatic calculi, and polycythemia vera was given an oral dose of 6 millicuries of radioactive phosphorus, and five months later a second dose of 10 millicuries, with reduction of hematocrit to normal levels. During the period of observation, the vesical calculus grew larger as observed roentgenographically and through the cystoscope. Two months after the second oral dose of radiophosphorus, the vesical calculus and some of the prostatic calculi were surgically removed. After washing for two days to remove surface urine radioactivity, the stones were placed before a Geiger counter. The vesical calculus was markedly radioactive. Autoradiographs of the bisected stone showed the radioactivity to be localized in a peripheral lamina, presumably that part of the enlarging stone which had been laid down while the patient's urine contained radiophosphorus. The prostatic calculi, which were not in contact with the urine, and which presumably were not growing during the period of radiophosphorus therapy, were not radioactive.—*Henry P. Brean, M.D.*

BOYD, GEORGE A., CASARETT, GEORGE W., ALTMAN, KURT I., NOONAN, THOMAS R., and SALOMON, KURT. Autoradiographs of C^{14} incorporated in individual blood cells. *Science*, Nov. 12, 1948, *108*, 529–531.

The individual cells of a rat's blood taken twenty-five hours after the intraperitoneal injection of glycine containing C^{14} in the alpha-carbon atom were smeared directly on a special emulsion, and after a sixty-seven day exposure were developed, stained and photomicrographed.

The identifiable cells were the lymphocytes, polymorphonuclear leukocytes and the erythrocytes. The autoradiographed cells showed varying concentration focally about them of a non-uniformly distributed silver grain. (The number of silver grains per unit area in the autoradiographs is a measure of the relative units of C^{14} incorporated in the cells.)

Most of the lymphocytes autoradiographed, the polymorphonuclear leukocytes occasionally and the erythrocytes rarely. This phenomenon probably lies in the rate of formation and the

relative amounts of nuclear proteins in the three types of cells.

The erythrocytes which autoradiographed apparently were newly formed indicating utilization of C^{14} in the bone marrow and not in the peripheral blood.—*William H. Pfisterer, M.D.*

MISCELLANEOUS

Mackay, Maurice W. H. A viscous diodone as a substitute for iodized oil. *Brit. J. Radiol.*, April, 1948, *21*, 204–205.

A water soluble solution consisting of 35 per cent of diodone with a varying percentage of polyvinyl alcohol as a thickening agent has been used as a contrast medium. Both substances are non-toxic in the amounts used and, in fact, the author, with others, submitted himself to intravenous injections of this material for proof. The sterilized solution is used primarily for uterosalpingography to replace iodized oil, obviating the danger of inadvertent introduction of the oil into a venous channel, and circumventing the slow absorption from the peritoneal cavity.

It has also been found excellent for injection of fistulous tracts, cystography, and even as a medium for examination of the mucosa of the pharynx and esophagus.

The material has been used for twelve months in amounts up to 60 cc. for one patient, and no untoward symptoms or signs have been noted.—*E. F. Lang. M.D.*

Howes, William E., and Alicandri, B. Bruce. A method of roentgenologic examination of the shoulder. *Radiology*, May, 1948, *50*, 569–580.

The authors present a study of the roentgen anatomy of the shoulder locating the sites of the facets of the short rotator muscle tendon insertions with lead foil. From the study, five views are suggested and illustrated with the purpose of (1) locating more accurately the sites of calcification within the tendon sheaths and in the bursa (2) clarifying the exact location of fractures of the tuberosities, and (3) identifying periosteal and subcortical zones of reaction secondary to tendon injury.

The article contains several good illustrations. The study of the roentgen anatomy in the five positions is worth referring to the original article itself.—*Frank J. Rigos, M.D.*

Donaldson, S. W., and Cheney, William D. Prenatal estimation of birth weight by pelvicephalometry. *Radiology*, May, 1948, *50*, 666–673.

The Ball technique was used in determining the size of the pelvis and the circumference and volume of the baby's head. It is concluded that the size of the fetal head is the best known index at the present time for estimating birth weight. Such factors as muscular development, skeletal size and fatty tissue may discredit the method to some extent, yet the error is not more than 10 per cent.—*C. H. Warfield, M.D.*

Kendig, Tom A. A simple pelvimeter to be used with the triangulation method of pelvimetry. *Radiology*, March, 1948, *50*, 395–399.

By means of an easily constructed plastic device, pelvic and fetal head measurements can be made directly from a lateral and an anteroposterior roentgenogram and corrected for distortion with the aid of an incorporated chart, thus increasing accuracy, saving time, and simplifying wet film readings.—*William N. Thomas, M.D.*

Jones, Glenn E., Grohowski, Alphonsus L., Robertson, Harold D., Ramsey, George H., Schilling, John A., and Strain, William H. Iodinated organic compounds as contrast media for radiographic diagnoses. *Radiology*, August, 1948, *51*, 225–236.

A comparison was made of four different iodine-organic compounds in regard to their visualization in biliary radicles and common duct in the dog. It was found that both standard gallbladder dye (priodax, for instance) and newer compounds of benzoic acid, delineated liver ducts when injected intravenously. The benzoic acid derivative appeared in the biliary system in ten minutes. When the various compounds were given by mouth, only the benzoic acid outlined the liver radicles.

The possible use of this compound in the study of post-cholecystectomy patients is suggested.—*Robert P. Barden, M.D.*

Nolan, James F., and Steele, James P. Carcinoma of the endometrium. *Radiology*, August, 1948, *51*, 166–176.

Report of experimental measurement by photographic method of radiation in gamma roentgens delivered to various shaped excised uteri. Under conditions of the experiment radi-

ation from multiple capsule implantations of radium are superior to the usual tandem method for the treatment of endometrial carcinoma. Details should be studied in the original paper.—*E. C. Baker, M.D.*

SETALA, KAI. Preliminary observations on the effects of irradiation upon the chylomicrons in human blood. *Radiology*, June, 1948, *50*, 803–810.

In order to further study radiation sickness chylomicron curves of 32 individuals were made. The chylomicron first used by Gage and Fish in 1924 is a method of studying the tiny droplets of fat visible in the blood plasma, after a fatty meal, in light reflected from their surface under the dark field microscope. By counting the number of chylomicrons at stated intervals following a certain method, after a fatty meal a so-called "chylomicron curve" can be constructed.

Since chronic administration of histamine is said to produce the same histopathologic picture as irradiation, including extensive accumulation of sudanophil fat around the central vessels of the liver lobules, histamine studies also were made. In order to eliminate possible errors the persons studied were kept under the same conditions during the whole observation period; the investigations were begun after a rest period of twelve hours; and the examination of the blood was always performed by the same person with the same instruments. It was found that the metabolic rhythm of the mobile visible blood lipids (chylomicron curve) was characteristic for each individual and its form for the same individual in same external conditions remained comparatively constant. The curve usually reached its maximum three hours after the fatty meal and fell to the 0-plane on an average of eight hours after the meal, practically irrespective of whether one or two fatty meals were taken provided the total amount taken was the same.

Irradiation given to persons considered healthy had a definite reducing effect on the mobile blood lipids, especially when given comparatively soon after a fatty meal, and to certain regions of the body. Irradiation of cancer-bearing patients produced similar changes in the amount and metabolic rhythm of blood lipids. In conformity with the clinical observations that the site of the irradiation has a bearing on the irradiation reaction, it was found that treatment over the pelvis, lower abdomen, liver, and the area of the bifurcation of the common carotid artery, caused distinctly greater changes both in the amount and in the absorption rhythm of the blood lipids. It was also demonstrated that the shorter the interval between the taking of fatty food and subsequent irradiation, the greater the effect of irradiation seemed to be.

The histamine studies showed that histamine also had a strong reducing effect on the amount of blood lipid. The effect was usually the greater, the sooner after the fatty meal the drug was administered. In some cases when given before the meal, it prevented the appearance of chylomicrons in the blood.

The author believes that irradiation causes definite changes in the metabolism of mobile visible lipids in human blood and also that changes of an exactly similar nature may be brought about by histamine acid phosphate administration. When the present observations are considered with work of other investigators it seems likely that irradiation and histamine administration lead to hemoconcentration when the permeability of the capillaries increases and the alterations in the amount of mobile visible blood lipids, especially their decrease, may be considered as partly attributable to the same cause. In other words, the mobile visible blood lipids escape more easily from the blood stream because of the increased permeability of the capillaries.—*Robert K. Arbuckle, M.D.*

FINK, KAY, and FINK, R. M. The formation of monoiodotyrosine from radioiodine in the thyroid of rat and man. *Science*, Oct. 1, 1948, *108*, 358–359.

The authors prepared two dimensional filter paper chromatograms and radioautographs of rat thyroid hydrolysates, following intravenous or intraperitoneal administration of 0.05 to 1 mc. of carrier-free I^{131}. Total dosage to the rat thyroids varied from 0.5 to 40,000 rep (roentgen equivalents, physical) of beta radiation, delivered at varying rates.

The chromatograms and radioautographs showed exactly corresponding radioactive spots with R_F values between tyrosine and diiodotyrosine, thought to be monoiodotyrosine, but not conclusively identified as such by crystal analysis.

A chromatogram of the hydrolysate from a biopsy specimen of a patient with thyroid adenoma, taken eight days after ingestion of

12 mc. of I^{131}, showed a radioactive spot corresponding to monoiodotyrosine.

Normal rat thyroids were similarly hydrolyzed in the presence of carrier-free I^{131} and only trifling amounts of monoiodotyrosine were identified, seeming to indicate that monoiodotyrosine is present in the irradiated thyroid before processing. The possibility is noted that iodine containing amino acids may be more easily broken down than are free amino acids.

Continuation of these studies should contribute materially to our increased knowledge of thyroid physiology.—*William S. Wallace, M.D.*

Patt, H. M., Swift, M. N., Tyree, E. B., and Straube, R. L. X irradiation of the hypophysectomized rat. *Science*, Oct. 29, 1948, *108*, 475–476.

Following lethal doses of roentgen radiation in the rat, there is an initial reduction in adrenal cholesterol, a secondary normal or increased value due to adrenal hypertrophy, followed by a marked terminal fall. The initial reduction can be prevented by administration of adrenal cortex, but the secondary rise and terminal fall are not affected. There is a similar fall in adrenal ascorbic acid following exposure to cold.

It is agreed that these adrenal changes are controlled by the adrenotropic hormone of the pituitary.

Sixty rats received total body irradiation with 750 r, generated at 200 kv.; half of these had been hypophysectomized seven days previously. The characteristic adrenal response to lethal irradiation was not seen in hypophysectomized animals, but the characteristic changes in spleen and thymus were not altered by pituitary extirpation.

Hypophysectomy seemed to enhance toxicity of the radiation, with almost half of the hypophysectomized rats dying within four days of irradiation. No rats with intact pituitary died in the first six days after irradiation and this group showed only 30 per cent mortality in sixteen days.

Hypophysectomy alone is known to produce adrenal atrophy and a drop in adrenal ascorbic acid.

Further studies are planned to determine whether the adrenal cortex is less sensitive to irradiation after hypophysectomy, due to removal of pituitary influence, or whether the adrenal stimulation after irradiation is entirely dependent on the adrenotropic hormone.—*William S. Wallace, M.D.*

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This article states the physical reasons why the skin tolerance is higher with supervoltages. The ionization produced in the surface region is both less than that available some distance below and composed of electrons of relatively higher average energy and therefore lower biological effectiveness. The increased skin tolerance must disappear almost completely when the radiation beam has traversed a distance below the surface equal to the range of the highest energy electrons which the beam can produce. The biological effect on the skin should be less the higher the voltage.—*C. H. Warfield, N. D.*

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The author reports the technique of 3 million volt roentgen therapy using a single port of entry 100 to 150 sq. cm. with a skin target distance of 100 cm., filtration equivalent to 2

mm. lead, 300 to 400 roentgens daily with the total dose up to 5,000 r.

The 10 cm. depth dose is 60 per cent, the skin erythema, both entrance and exit, is not visible and the systemic reaction on the part of the patient is less. This procedure is used in deep-seated lesions, cervical and buccal lesions, but not breast lesions.—*C. H. Warfield, M.D.*

LEVIN, MORTON L. Some epidemiological features of cancer. *Cancer*, Sept., 1948, *1*, 489–497.

The increase in cancer mortality has been attributed to improved diagnosis and "aging" of the population. The author shows that the increase has stopped since 1920 in England and Wales and since 1935 in Massachusetts where the data are standardized for age. He also points out that the major increase in mortality took place between 1850 and 1895 prior to the discovery of roentgen rays.

There has been a marked sex difference, with females showing the greatest cancer mortality, until 1920 in England and Wales and 1935 in Massachusetts at which time the rates in males exceeded those in females. This difference, the author believes, is due to improved diagnosis in both males and females which has been counterbalanced in females by improved cure rates.

The author lists forty-five statistically significant precancerous lesions and briefly discusses such epidemiological factors as age, socio-economic position, carcinogenic chemicals, radiation, and hereditary factors.—*W. E. Childs, M.D.*

SIMON, SAMUEL. Sudden death following intravenous administration of diodrast. *J.A.M.A.*, Sept. 11, 1948, *138*, 127–128.

The basis for this report is a fatality subsequent to the intravenous administration of iodopyracet injection ("diodrast"). The author reviews some of the literature; he states that the incidence figured 1 fatality to 25,000 injections. Two types of death were reported: (1) immediate, due to allergy to the drug, (2) delayed due to pre-existing major renal damage.

He reports a case and states that the clinical impression was that death was of allergic type. On the basis of the postmortem observation, the following precautions were listed and discussed: (1) question patient carefully as to allergy; (2) cutaneous tests performed; (3) oral test performed; (4) drug administered slowly; (5) emergency tray kept available; (6) non-protein nitrogen or urea nitrogen on patient prior to pyelogram; (7) increased danger of jaundiced patients; (8) danger of repeated urographic examinations. A review of the literature showing advantages of these precautions is given.—*Robert D. Moreton, M.D.*